THE JOURNAL
of
SPEECH DISORDERS

Volume 9 SEPTEMBER, 1944 Number 3

WAR APHONIA

RALPH R. SOKOLOWSKY, M.D.[1]
Capital University, Columbus, Ohio
in collaboration with
MAJOR E. B. JUNKERMANN, M.C.
Station Hospital, Camp Ellis, Illinois

I.

During the first World War there occurred a striking number of cases of psychogenic aphonia.[2] The publications from almost all the nations involved in the conflict confirm this statement.[3] While this condition occasionally comes to the attention of the physician in peace-time,[4] such sporadic cases appear almost exclusively in women with a more or less obvious neurotic background. In contrast to this, there appeared in a constantly increasing number, during the first World War, cases in healthy young soldiers who often displayed no other hysterical stigmata. This fact, as can be readily understood, aroused the interest of the laryngologist, the psychiatrist, and the speech therapist.

This interest, however, is more than a strictly scientific one. The great number of cases which occurred compelled the military authorities to give the problem their serious attention. This was evidenced by the fact that the author himself had the opportunity to examine and treat 116 cases of psychogenic aphonia during the first World War. It is a reasonable conclusion that if 116 cases came to the attention of one physician, the total number of such disturbances must have been very

[1]Dr. Sokolowsky's death occurred June 25, 1944. Inquiry addressed to him concerning the documentation of the sources mentioned in footnote 4, and of items 19(b), 22, 23 and 28 in the References at the end of this article, failed to reach him before his death. A brief tribute to Dr. Sokolowsky, prepared by Professor W. C. Craig, appears in the News and Announcements section of this issue of the *Journal*.

[2]The term *aphonia* may be defined briefly as "the loss of voice," so that speech is reduced to a whisper.

[3]In the writings published in the United States, at least so far as has come to the attention of the author, only one reference has been found alluding to aphonia in war. Colonel Richardson (21) reported among 37 speech patients only two cases of aphonia, along with 14 aphasics, 13 cases of imperfect phonation, etc.

[4]Even after the last war, up to 1941 we find in the literature reports on one to three cases of psychogenic aphonia: Falcao, 1926; Skvortzoff, 1927; Oertel, 1928; Barczinski, Torres, 1932; Minimitani, 1938; Miyta, 1941; etc.

great. It is probable that many similar cases will occur in the present conflict among both the soldiers and the non-combatants because of the hitherto unknown violence of this total struggle.

The publication of the experience gained in dealing with such a large number of aphonias during the last war (22) seems to be justified at this time in order to avoid a repetition of our initial disappointments and unsatisfactory therapeutic procedures. Ultimately, we were able to achieve almost 100 per cent restoration of the voice. Moreover, in this article, our gradually acquired knowledge and experience will be correlated with the results of investigations made during the intervening years. It will, however, be readily recognized that the peculiar conditions present in war aphonia are not comparable to those surrounding the condition in peace time.

Almost all the publications concerning the psychogenic aphonias in the first World War were based upon observations made in base hospitals usually situated hundreds of miles back of the front line in the interior of the patient's native country. In contrast, our special otolaryngological station was a section of a front line hospital or even of a field station. In the latter case, the aphonics came directly from the combat organization to our station. This fact points to at least one very clear difference; our cases were generally "recent," and had therefore received very little or no treatment, while in the remote base hospitals the cases were received after a much longer interval. It will be shown that this fact is significant both in regard to the therapy and to the prognosis. In addition, our special station was active in the more quiet sectors of the eastern front as well as in the sectors where the fighting was violent on the western front. This difference in the origin of the cases was of special interest in the etiology of war aphonias.

II. Etiology

Psychogenic or functional aphonia is, according to the majority of authors, a hysterical disturbance. In accepting this view we must, however, be fully cognizant of the fact that *psychogenic* and *hysterical* are not identical terms, and that, properly stated, the hysterical reaction is but a part of the psychogenic reaction. Every hysterical symptom may thus be considered a psychogenic one, but not every psychogenic symptom may be regarded as a hysterical one. The assumption of the condition being a hysterical disturbance does not contradict the fact that in our psychogenic aphonias we frequently found no other hysterical stigmata (monosymptomatic form of hysteria).

Some authors desired a special classification. Isserlin and Nadoleczny (19) suggested that they be differentiated into two types: (1) thymogene aphonia resulting from a fright, usually

rather trifling, a slight accident, or even following a spell of listlessness; (2) ideagene aphonia caused by anxiety (apprehensions), pangs of conscience or by hypochondria. Obviously the latter type may be more serious and deep-rooted, hence more resistant to treatment. McLeod, cited by Lell (13), proposed a classification into (1) true aphonia, where there is little or no approximation of the vocal cords in phonation; (2) hyperphonia, where the vocal cords are brought forcibly together and the ventricular bands are also approximated, making phonation almost entirely impossible. Lell also cited the view of other observers who preferred to classify functional aphonia as voluntary (the condition of the malingerer), and involuntary(true cases of functional aphonia). Marsh (16) found it advisable to classify functional aphonia into (1) a primary class exemplified by the hysterical cases and (2) a secondary class exemplified by the cases that start with laryngitis secondary to a septic focus in the upper air passages. For practical purposes we preferred to combine all these forms under the common name, *war aphonia.*

It was, of course, very tempting to bring psychogenic aphonia into relationship with traumatic neurosis, since not infrequently in the various articles a trauma was given as the cause of the aphonia. Explosions of shells and mines, and particularly being covered with debris due to explosions, were frequently stated to be the causes of aphonia. Muck (18) found 13 among 20, Fröschels (6) 14 among 70, Marx (17) 4 among 13 of aphonias to have been caused by explosions and being buried by debris. Winslow (28) mentioned simply "exposure to incessant gunfire" as an important cause of functional aphonia. Other incidents were stated anamnestically, as hoof-kicks to the chest or bullet wounds, and in addition comparatively slighter injuries, such as a blow to the shoulder by the tail end of the gun carriage, or even the administration of politzerization or vaccination.

In contrast, other authors had among their aphonias only a small number caused by shell or mine explosions or even by slight injuries. Hence any connection of the war aphonia with traumatic neurosis must be dismissed. In this respect the author's cases are still more distinctive and marked. As long as our special station was active on the more quiet eastern front, 82 aphonias came under our observation. Among these cases there was not one whose disturbance was the result of the above mentioned severe or even slight traumas. We ascribed this at first somewhat unexpected fact to the minor fighting activity in our front sector. All the more were we surprised when subsequently, under absolutely changed conditions, in a sector of severe and violent fighting on the western front, we met similar anamnestic reports. Here another 34 aphonics came

THE UNIVERSITY OF MICHIGAN LIBRARIES

under observation within a few months. Even of these aphonics none attributed his disturbance to the above mentioned grave war accidents.

In general the majority of the aphonics ascribed their loss of voice to a "cold" caught in the ordinary course of military life, such as guard duty, living in wet trenches and dugouts, prolonged marching in bad weather, etc. Not infrequently the aphonia originated in the rest quarters. A larger group of aphonics came from a recruit depot in the quiet front sector; in other words, aphonia occurred in recently-called youthful individuals who had not yet participated in any fighting. In many cases the origin of the aphonia was ascribed to a preceding "sore throat." Typical for a great number of cases, particularly in the quiet sector, was the following current anamnesis: the patient was on guard duty at night exposed to a high wind, the next morning he felt a "sore throat," and on the following morning he awoke completely voiceless. In the front sector with more fighting activity several cases were attributed to the inhalation of poison gas. However the laryngoscopic examination displayed no difference between these cases and the other aphonias.

From the cases that came under our observation it thus became fully apparent, first, that the war aphonias did not require any grave trauma for their appearance, and second, that the majority of the patients ascribed their aphonia to a "cold," a "sore throat" or a "laryngitis." In fact, the examination showed, in a number of cases, definite findings of laryngitis. Some of them had also suffered previously from "catarrh" and "hoarseness." It was not surprising, therefore, that such individuals easily developed aphonia following their hoarseness. Quite frequently such cases have been found to be so called "sparing aphonias" (the aphonias of non-use, *Schonungsaphonie*). The thought of making their "laryngitis" or their "cold" worse, or the fear of losing their voice entirely, compelled such patients unconsciously to spare the voice as much as possible and thus finally to speak only in a whisper. In the emotional life *(Gefühlsleben)* of such individuals the voice plays such a prominent part that they react to ordinary organic transformations of the larynx in this severe form.

There remained, however, a considerable number of aphonics who, it is true, ascribed their voice disturbance to a "cold" but whose laryngoscopic findings were absolutely negative. To be sure, in wartime a soldier is subject to many conditions which may cause him to contract a cold. Hence it is quite logical for the aphonic to attribute his "hoarseness" to such a "cold." With a number of these aphonics we could take a hysterical disposition for granted, particularly since, due to the long duration of the war, there was no longer the initial "cream" of man-

power in the army. This was true with respect to the physical fitness as well as to the *psychical power of resistance.* This was proved by the fact that almost all of the authors in question, including the present author, could reveal a continual increase of psychogenic aphonias during the war. This latter part of our aphonias etiologically showed a distinct parallel with the peace-time aphonias as recently described by Greene (9): "The patient almost invariably lost the voice following a 'cold' . . . . the physcial disfunction is due to neurotic background and psychical influence." It stands to reason that the majority of the writers stressed at all times the emotional and social background in peace-time aphonia, whereas other writers such as Mackenzie (15) and recently Lell (13) emphasized that the most outstanding etiological factor in peace-time aphonia is an acute laryngitis.

To recognize the onset of war aphonia, however, it is not always necessary to assume the existence of a hysterical disposition. In many cases the aphonia was the expression of an "abnormal reaction" (*Abnorme Reactionswcise*) to the completely changed and extremely demanding requirements of military life in wartime, which, as we have seen, need not necessarily be spent in trenches or even in the front line (casual hysterics, *Gelegenheitshysteriker*). Quite frequently it concerned men who had previously lived under simple and quiet conditions in the rural districts, who were suddenly separated from their families and found themselves in military training with its unconditional subordination, where they had to live with many other soldiers and experience the resulting frictions and irregularities of military life. In addition there were the severe strains brought about by the war service with its greatly increased demands upon the individual. To a certain degree the individual could stand up under these havoc-producing conditions; this maximum being reached, it often needed but a minor event to bring about the breakdown of his resistance. This event was frequently so insignificant that it could scarcely be recognized or was completely missed in the anamnesis. We thus learned also that the male psyche, given a certain degree of emotional stress, showed a surprising tendency for functional aphonia.

Of a certain importance, also, as a source of war aphonia were the "ideas of wishful desires" (*Begehrungsvorstellungen*). In this respect we had come to the realization that these ideas were not so apparent with the *origin* of the aphonia as with the *fixation* of the already present disturbance. Hence the aforementioned "ideas of wishful desires" were scarcely etiologically important with our cases, since throughout they were largely acute and untreated. In contrast, these ideas played an essential part with the aphonias in the remote base hospitals in

THE UNIVERSITY OF MICHIGAN LIBRARIES

the home land, where the ease of hospital life and particularly the influence of other hospital inmates created the atmosphere that Pflug had strikingly designated as "hospital breeding" (*Lazarettzüchtung*).

On this point we could also realize an essential difference between our cases and the aphonias which came under observation in the base hospitals remote from the front lines. This difference became fully apparent, as we shall see, in the results of the therapy.

It may be worth mentioning that war aphonia sometimes occurred epidemically. Five such cases came under our observation. In a training camp in a quiet sector on the eastern front four recruits of the same barrack became aphonic, one after the other. Further, a bed patient with sinusitis who was in our hospital station close to a newly admitted aphonic reported at the medical visit next morning completely voiceless.

III. Diagnosis

In general the diagnosis was not a matter of great difficulty, particularly as the war progressed and we became more familiar with this disease. The majority of diagnostic errors occurred in those cases where the laryngoscopic findings revealed distinct symptoms of laryngitis. The typical light thickenings of the mucous membrane in the region of the arytenoids or the anterior commissure, or the color changes of the cords, dominated the clinical picture in certain cases to such a degree that even outstanding laryngologists were mistaken and contented themselves with the diagnosis "laryngitis." In this regard the following occurrence was very characteristic. At the special station of one of the most outstanding phoniatrician-laryngologists, for whom I had to substitute for several weeks, I found two aphonias considered, and therefore treated, as laryngitis. The evidence that the two cases were actually aphonias was shown by the fact that both patients readily regained their voice with but a single treatment.

In the majority of the aphonias which were combined with laryngitic symptoms, the laryngitis, as the anamnesis revealed, was the primary etiologic factor. On the basis of this laryngitis the aphonia then developed, quite frequently under the form of the aforementioned "sparing aphonia." The course of these cases confirmed clearly that the laryngitis was really the primary and causative etiologic factor of the aphonia. After the return of the voice there often remained a huskiness or roughness, as is ordinarily found in laryngitis, which quickly disappeared under the usual laryngological treatment. The following consideration proved helpful with the differential diagnosis: We know that a genuine *organic* total aphonia (i.e., total loss of voice) is a comparatively rare event. We will find it only in cases of true paralysis of the cords or under the most serious

organic transformations, such as the gravest ulcerous destructions, intrinsic growths, etc. In contrast, in the cases of laryngitis, even the most severe, the patient will almost always be able to produce a sound by request. To be sure, the production of this sound will require more or less effort on the part of patient; in addition, this sound will be more or less of a rough, husky, grating, hoarse quality. But it will not be merely a whisper. Consequently we came to the following conclusion: If an aphonic with distinct laryngitic symptoms insisted that he was unable, even wth the greatest effort, to produce a sound, and kept firmly to his whispering voice, we felt at liberty to suppose that a functional aphonia was superimposed upon the patient's laryngitis. The results of the immediate therapeutic procedings, directed toward the aphonia and not toward the laryngitis, proved our point.

On the other hand there were, to be sure, rarer cases where the laryngitic symptoms occurred secondary to the aphonia. We were familiar with these findings from the etiology of phonasthenia (the functional voice disturbances of professional speakers and singers). In phonasthenia the voice is not produced by a *minimum* power, but by a much greater exertion on the delicate laryngeal muscles than would be necessary to produce the intended tone effect. This misuse of the vocal tract leads finally to inflammatory conditions in the larynx. We found very similar pictures in the larynges of the aphonics as a consequence of an over-intense and too greatly forced phonation.

Most striking, however, in the laryngoscopic picture were the disturbances in the movements of the cords. These disorders were manifold. An entire range of such disturbances came under our observation, starting with a complete relaxation of the glottis, as in the paralysis of all adductors, and extending to a cramped closing of the glottis, where the closed ventricular bands scarcely permitted a view of the cords. Between these two extremes (hypokinesis and hyperkinesis) there were to be found pictures which resembled more a paresis of the internal thyro-arytenoid, or of the transverse arytenoid muscles, or a combination of both forms. A peculiar laryngoscopic picture appeared in those cases where the cords approached each other in the midline under certain 'atactic" movements without completely coming together. The latter form seems to have dominated the clinical picture in the aforementioned peace-time aphonias of Lell (13).

Quite frequently the laryngoscope revealed a spastic closing of the ventricular bands in their fore and middle third while through the open rear third the typical triangular gap of a paralysis of the transverse arytenoid muscles was visible. Functionally, in some cases this form did not result in a genu-

THE UNIVERSITY OF MICHIGAN LIBRARIES

ine aphonia but in choked, husky voice, the so-called "ventricular-bands-speech" (*Taschenbandsprache*). In all our 116 cases we found but six ventricular-bands-speakers. Seemingly other authors found them more frequently, as Kessel (12) with war aphonias and MacLeod, cited by Lell (13) with peace-time aphonias. Isolated paralyses of the lateral crico-arytenoid or of the cricothyroid muscle, as seen by Amersbach (1) in one case, did not come under our observation.

In all our cases showing any disturbance of movement of the cords, *both* cords were equally afflicted. Hence we were inclined to consider the bilateral affection as significant and typical for psychogenic aphonia in spite of the isolated case of unilateral disturbance observed by Beck in wartime.

The continuous change of the laryngeal image with the same patient was also typical in many cases. We found this variability, this effacement of the borders, this transition from one form to another (even from the spastic to the paralytic form) not only with examinations carried out in different ways, but also during the same examination, provided it could be sufficiently prolonged.

Anomalies of breathing were found quite frequently. Nadoleczny (19) had examined the breathing objectively, recording the respiratory movements by means of a pneumograph. In the main he revealed a polypnoea, an increase of the frequency of the inspirations. With our patients—observed, of course, without any apparatus—we found as a characteristic great expenditure of the expiratory air, a marked "phonatoric waste of breath." The greater the paresis of the adductors the more pronounced was the expenditure of expiratory air.

IV. Therapy

Our therapeutic procedures with war aphonia had to undergo several changes during the war. These changes were, in the main, due to our not being completely satisfied with the results obtained; we therefore set about to improve our therapeutic measures. Moreover, in the last year of the war, especially from the spring to the fall of 1918, another change in our therapeutic procedure took place. This was caused by external circumstances. On the more quiet eastern front all our aphonics had been hospitalized, while after our transfer to the sections of violent fighting in the west we were forced to treat the majority of our aphonics in the outpatient department. In the daily much-frequented special clinic the treatment, naturally, could not be administered so painstakingly as in the case of the hospitalized patients in the east. But in spite of that our therapeutic results improved continually. We shall learn shortly the reason for this rather strange fact.

Like the majority of the authors who had to deal with the treatment of war aphonia, we first introduced that therapy with

which we were familiar in time of peace, phoniatric reeducation. We administered breathing exercises in connection with a systematic speech and voice training. This orthophonic treatment of war aphonia has been varied by different therapeutists. Curschmann (4) made successful use of the principles of the singing method of Hey, based on a humming tone; Guttmann (10) referred to the directions of the voice therapist, Theodor Paul; Garel (7) had the patient make a forcible expiration, like a sigh, having him, further, to make this expiration audible with a sound. As an expedient he suggested a sudden pressure on the epigastrium with one hand, the other hand on the back. After the war, Dundas-Grant (5) recommended the "inspiratory phonation of the vowel sound 'ee' followed by the expiratory 'aw'." As an auxiliary help he had the patient press together the hands of a nurse standing behind him. This procedure is based on the observation of Negus that in certain animals the efforts made with the fore limbs were accompanied by an approximation of the vocal cords.

By means of the phonetic reeducation we succeeded in restoring the voice to about 60 per cent of our aphonics, a rather meager result considering the relatively tiresome treatment which sometimes lasted several weeks. The best results with the orthophonic treatment were apparently obtained by Garel (7) (about 70 per cent cured).

After all, it is obvious that all treatment of the aphonia, including the orthophonic treatment, can *a priori* be but a suggestive one. The crucial point of the entire phonetic reeducation was the moment immediately after preparatory breathing exercises where the aphonic had to be led from whispering to the vocally pronounced voice. Once a sound had been achieved, the further completion of the cure usually did not meet any difficulties. But on just that critical point hinged our successes or our failures. We had to conclude that in our failures the mild orthophonic treatment was not efficient enough to liberate the patient from his idea that he had an established disease.

Induced by the publications of Muck (18) and his extraordinary results we then tried his method—the introduction of a pellet or a small ball into the larynx between the vocal cords, in order to bring about a sensation of being suffocated which superinduced a cry of fright. We have to confess that our own results with Muck's ball were not very encouraging. We were, however, fully aware that our failures did not discredit the method per se. In his article, "Concerning the Psychically Caused Disturbances of Hearing in Soldiers," Kuemmel says, "What cures him is not the method but the physician who employs this method." We can easily understand that with a disease where the entire result depends on the personality of the

therapist the same method shows excellent results in the hands of the one while it is rather inefficient when employed by another.

In the post-war literature we find several methods reported for the treatment of peace-time aphonias which, although entirely different, are based on a principle similar to Muck's. Winslow (28) and subsequently MacMahon, cited by Lell (13), introduce a finger into the patient's throat. MacMahon depresses the back of the tongue and with it the larynx firmly; at the same time the other hand presses the posterior edges of the thyroid cartilage firmly together. Winslow lowers his fingers beyond the epiglottis. It is obvious that in both procedures the finger plays, *mutatis mutandis*, a similar role to Muck's pellet, bringing about an uncomfortable, uneasy sensation which finally leads to a form of vocally produced voice.

Moore, cited by Lell (13), and particularly Lell, ardently advocate the direct laryngoscopic inspection. According to Lell the anterior commissure laryngoscope is the instrument of choice. The tip of the laryngoscope is introduced between the ventricular bands; when the patient, by request, attempts to phonate "there is sufficient spasm of the true cords from the pressure of the laryngoscope to bring the cords together." Fully acknowledging the explanation of Lell the writer wishes, in addition, to call attention to certain uneasiness more or less with each bronchoscopic inspection, and particularly to the peculiar, unnatural, and somewhat helpless attitude in which the patient is requested to phonate while the bronchoscope is lying in the larynx. We will readily realize that at least some of the particulars that are important with the procedures of Muck, Winslow, etc., are also effective with the bronchoscopic inspection.

We achieved far better results with the administration of a short open drop anaesthesia combined with a feigned manipulation, a subcutaneous injection of saline solution into the neck region. With this method, introduced by Goldstein, we were able to succeed in a number of those cases where we failed with the orthophonic treatment. The patient was told that to regain his voice he had to receive an injection which would, however, be very painful, hence the narcotization. At the very moment that the patient came out of the anaesthesia he was urged to speak. As a rule this could easily be carried out quite successfully with the still benumbed and not fully conscious man. It was, however, absolutely necessary to continue this "question and answer play" until the patient became fully aware of the complete restoration of his voice. In one particular case the success we had obtained was lost again because we stopped too soon with the question and answer parlance. In another case the sensation of being suffocated brought about by the mere place-

ment of the narcosis mask was sufficient to cause the cry of fright. This cry we used, of course, after having removed the mask, immediately for the full development of the voice. The first inhalations of ether in this case worked, *mutatis mutandis*, like Muck's pellet.

From the very beginning of our experience with the war aphonia sporadic cases came under our observation where the mere introduction of a laryngoscope mirror sufficed to restore the voice, patients who were, so to say, but waiting to be "redeemed." From this type of cure came of itself the effort to develop the voice during the first examination, a prolongation of the time for the introduction of the laryngoscopic mirror, and putting the mirror somewhat deeper into the throat in order to get a vocal retching reaction. All of this was reinforced by verbal suggestions.

After being successful in effecting a cure by this procedure in a number of relatively more obstinate cases we gradually became so familiar with this very simple and really very humane form of "surprise attack" that in the last months of our practice at the quiet eastern front we had scarcely a failure. Hence we subsequently tried the same procedure in the section of violent fighting in the west. Because of existing conditions the majority of our aphonics (22 out of 34) were not hospitalized patients but were treated in our outpatient department. We had only one room at our disposal where another surgeon and the author had their desks close together and attended their numerous patients during the same hours. In addition, a number of patients were waiting for treatment in the same room. All of these unfavorable circumstances, however, did not impair the results, provided the procedure was followed along energetically and quickly.

As soon as the anamnesis pointed to a psychogenic aphonia and the laryngoscopic mirror confirmed this assumption a short remark such as, "You will be all right quite soon," or, "You will be getting your voice back quite soon," was made. After that the patient was not permitted, so to speak, to "collect his wits." All manipulations such as setting the head in proper position and pulling out the tongue were carried out as quickly as possible, and accompanied by short, crisp and somewhat commanding words. Then followed the deep introduction of the mirror and the attempt to obtain a vocal retching reaction. After this was accomplished, it was brought into the consciousness of the patient with short, crisp remarks, such as, "Here we are," "Now your voice is back," or, "Do you hear your voice?" After that it required but relatively little effort (the mirror, of course, remaining continually in the throat) to elicit from the patient the unpleasant gag-reminding but nevertheless audible "ah." The further development, of course, during

the same treatment and always in the aforementioned concise, urging, definite and exacting form, usually did not meet with particular difficulties. In order to secure the full measure of success the patient was referred to a nurse, accomplished in this field, who had the patient continue the exercises, counting, reading aloud for some time.

A few particularly obstinate cases, especially where a vocal retching reaction could not be obtained, required still other minor help, such as the compression of the thyroid cartilage. This manipulation, especially when purposely administered with more firmness, acted not only by way of a mechanical approach of the vocal cords but also through the unpleasant sensation. This compression in connection with the growing urgency of the verbal suggestion led finally toward the desired result.

We considered it of utmost importance to avoid even the slightest intermission during the treatment. The frequent requests of the patients for a rest were disregarded; the entire treatment had to be accomplished from beginning to end without interruption. It cannot be overemphasized *that the complete restoration of the voice in the first and only sitting carried out continuously without interruption was the keystone of the entire procedure.*

As previously stated, out of the 34 aphonics that came under our observation in the sector of violent fighting in the west during the last months of the war, there were but 12 hospitalized patients. Eleven of these were cured during a first and only treatment, but we were unsuccessful with the twelfth. This single failure was due to the author's not feeling well on that particular day, so that he was unable to summon up the necessary energy. This twelfth patient, however, later regained his voice by administration of the aforementioned short open drop anaesthesia.

Out of the 22 aphonics treated in the outpatient department, 21 were cured with the first treatment; with only one were we unsuccessful. In this instance we could foresee the failure, as the patient brought with him an unsealed message from his regimental surgeon, inquiring whether the patient would presumably return to his front line duty or would be designated for "behind the lines" work. It was *a priori* rather likely that this question, thus made known to the patient, would unfavorably influence the result of our therapy. This was a rare but distinct instance of an iatrogenic (caused by physician) fixation of functional aphonia leading to a real flight into disease.

The author was fully aware that his way of proceeding did not represent an entirely new "method" of treatment of war aphonia. On the contrary, he was convinced that some of his

colleagues, who had to deal with aphonics, had also effected occasional cures in the same or in a slightly different way. Actually Garel (7) reported some cases where the mere introduction of the laryngoscope restored the voice.

Seemingly there were two reasons why we were finally able to achieve such an extraordinarily large number of cures (nearly 100 per cent) in spite of unfavorable extrinsic circumstances: First, in the course of time we gradually became more familiar with this procedure and attained a certain perfection of technique, and secondly and chiefly, the great majority of our cases came directly from the combatant troops and were therefore "recent." They had not been previously treated nor had they been exposed to the unfavorable influence of the remote base hospital in the home land nor to the detrimental effect of "hospitalism" *(Lazarettzüchtung)*. They were, therefore, far more susceptible to our treatment. After having realized the significance of the latter fact we reported our findings to our superiors with the suggestion that they call the attention of the surgeons to war aphonia, and that they refer all aphonics of our particular army to our station as quickly as possible instead of sending them to the distant hospitals far back of the front line or even in the home country. We did not succeed with this, however, because the general retreat which led to the Armistice began shortly.

There are many and weighty reasons for creating a center for the correction of speech defects far back from the front in foreign countries, or even in the United States, as was so successfully carried out in the first World War under the direction of Colonel C. W. Richardson at General Hospital 11 at Cape May, New Jersey. In such centers the correction and reeducation of speech defects can be carried on under far more favorable circumstances. The functional aphonias, however, should be exempted. It is apparent that since immediacy is the primary factor in the prognosis of such war aphonia, it is inadvisable to transport such a case either to an interior district or back to the United States prior to the treatment which has been described.

The described procedure is as simple and effective as the different "shock" treatments but certainly more humane than these, particularly the distressing faradic stimulation. This latter the author could not personally bring himself to accept, although at that time it was greatly recommended and carried out with war neuroses. The writer was pleased to find subsequently that a number of authors shared this dislike for the faradic stimulation in cases of functional aphonia: Dundas-Grant (5), MacMahon (cited in 13), Lell (13).

V. Recidivism (Relapses)

Many authors emphasize the great tendency to relapse with war, as well as with peace-time, aphonias. From the cases

that came under our observation this fact could only be partially confirmed, as only 10 of our 116 aphonics declared that they had already been voiceless previously. To be sure, we could not say what became of our recently cured cases which had been sent back to their troop units immediately after restoration of their voice. This was especially true with our 34 patients in the western theater of the war with its violent fighting, where the entire situation involved a continuous shifting of the position of the troops. On the eastern front it was, of course, with reservation, somewhat easier to form an opinion because there the units usually remained for months and even for years with the same army, so that the patients returned again and again to our station. While some patients, for example those with recurrent purulent otitis, returned to our station as often as 4 or 5 times, we met only two of our cured aphonics. After all, we do not want to draw any definite conclusions from this fact.

VI. Malingering

It was not always easy to determine the part played by malingering in war aphonia. Since it was a psychogenic disorder, it was sometimes quite difficult to draw a clear borderline between "the conscious" and "the unconscious" (*Bewussten und Unbewussten).* There are many connections and transitions between the unconscious desire to escape being cured and conscious malingering. We found this view subsequently confirmed by Winklemann (27), in peace-time aphonias.

In the great majority of our aphonics we had the impression that there was no malingering present. This was evidenced by the numerous assertions of happiness and gratitude for the restored voice. The base hospitals far back from the front line, with their comforts and the resulting "ideas of wishful desires" *(Begehrungsvorstellungen)* and the "desire to be considered ill" (*Wunsch zur Kranheit, Bonhoeffer*), seemed to be far more favorable for creating malingering. We refer to Lewandowski and Singer (14), who at first, according to their experiences in the field, denied the existence of malingering in war neuroses. Subsequently, however, from their observations in the base hospitals, they could no longer uphold their former statements. Here again we could see the unfavorable influence of remote hospitals on the war aphonias.

VII. Summary

1. The first World War displayed a striking frequency of psychogenic aphonias ("war aphonia"). One hundred sixteen cases came under the observation of the author.

2. Because of the great number of war aphonias they required the urgent attention of military authorities.

3. There is the probability of the recurrence of many cases in the present war among both soldiers and non-combatants.

4. A publication of the experience gained on 116 cases of war aphonia seems to be justified at this time, in order to avoid, in the present conflict, the faults of our initial unsatisfactory experiments, which we were gradually able to overcome, and instead of which we were able to perfect a procedure which resulted in almost one hundred per cent cures at that time.

5. Our final achievement was due not only to the gradual perfection of our "technique," but chiefly to the fact that we gained our experience in front line hospitals as well as in field stations, and not in a base hospital in the interior districts of the home country, on experience in which the other publications concerning war aphonias were based. Hence our cases were "recent," and had not as yet been treated nor exposed to the unfavorable influence of the base hospital in the home land. They were therefore far more susceptible to our treatment.

6. It is therefore inadvisable to transport an aphonic to a speech correction center which is far removed from the front (although these centers are invaluable for the correction and reeducation of all other speech defects, particularly the aphasias). Each aphonic should, however, be sent immediately to a station directly in the army zone for adequate treatment.

References

1. Amersbach, Karl. Erfahrungen über funktionelle Larynxstörungen bei Heeresangehörigen. *Arch. Laryngol.*, 1916-1918, **30**, 139-170.
2. Babbett, J. A. Aspects of aphonia, psychic and otherwise. *Ann. Otol., Rhinol. & Laryngol.*, 1923, **32**, 879-885.
3. Barlow, R. A. Newer concept of hysterical aphonia. *Tr. Am. Laryng. A.*, 1930, **52**, 23-34.
4. Curschmann, H. Bemerkungen zur Behandlung hysterischer stimmstörungen. *München. med. Wchnschr.*, 1916, lxiii, 1644.
5. Dundas-Grant, J. Aphonia, dysphonia, and the "singer's attitude." *Brit. med. J.*, 1930, **1**, 539.
6. Frœschels, Emil. Dei sprachärztliche Therapie im Kriege. II. Teil. Dysarthrien, traumatische Taubstummheit, Mutismen, hysterische Aphasien. *Monatsschr. Ohrenheilk.*, 1918, **52**, 385-423.
7. Garel, J. Treatment of war aphonia. *Lyon méd.*, 1919, **128**, 129.
8. Gold, L. H., and Garofalo, M. L. Treatment of aphonia by intravenous administration of pentothal sodium. *Anesthesiology*, 1940, **1**, 94.
9. Greene, J. S. (a) Psychiatric therapy for dysphonia: aphonia; psycho-phonasthenia; falsetto. *Arch. Otol.*, 1938, **28**, 213-221.
   (b) Psychiatric therapy in dysphemia and dysphonia: stuttering, psychophonasthenia, aphonia, falsetto. *Ann. Otol., Rhinol. & Laryngol.*, 1938, **47**, 615-631.
10. Guttmann, A. Stimmstörungen im Felde. Zur Technik der Uebungstherapie. *Dtsch. med. Wchnschr.*, 1918, **44**, 883-884.
11. Howard, C. N. Functional aphonia cured under general anesthetic. *J. Amer. med. Ass.*, 1923, **80**, 104.
12. Kessel, O. G. Taschenbandsprecher. *Z. Ohrenheilk.*, 1916, **74**, 69-71.
13. Lell, W. A. Diagnosis and direct laryngoscopic treatment of functional aphonia. *Arch. Otol.*, 1941, **34**, 141-149.
14. Lewandowski, M. Was kann in der Behandlung und Beurteilung der Kriegsneurosen erreicht werden? *München. med. Wchnschr.*, 1917, **64**, 989-991 and 1028-1031.

15. Mackenzie, M. A manual of diseases of the throat and nose. New York: Wm. Wood & Co., 1880.
16. Marsh, F. D. Functional aphonia; some observations on its clinical aspects. *Lancet*, 1932, **2**, 289.
17. Marx, S. Ueber psychogene Stimmstörungen bei Kriegsteilnehmern. *Beitr. z. Anat. Physiol., Pathol., u. Therap. d. Ohres* (etc.), Berlin, 1916, viii, 318-326.
18. Muck, O. (a) Beitrag zur Symptomatologie und Therapie der funktionellen Stimmstörungen im Kriege. *Z. Ohrenheilk.*, 1916, **74**, 139-153.
(b) Laryngeal pellet in treatment of aphonia. *J. Amer. med. Ass.*, 1921, **76**, 1437.
19. Nadoleczny, M. (a) Ueber funktionelle Stimmstörungen im Heeresdienst. *Arch. Laryngol.*, 1916-1918, **31**, 347-376.
(b) Handbuch Denker-Kahler, 5, 1929.*
(c) Was muss der Hals-Nasen-Ohrenarzt von Sprach — und Stimmheilkunde wissen? *Z. Hals-, Nasen- u. Ohrenh.*, 1938, **44**, 1-74.
20. Oaks, L. W., Merrill, H. G., and Oaks, L. E. Recurrent hoarseness and aphonia without demonstrable laryngeal pathology. *Ann. Otol., Rhinol. & Laryngol.*, 1932, **41**, 1079-1086.
21. Richardson, C. W. (a) Organization of section of defects of hearing and speech. *Ann. Otol., Rhinol. & Laryngol.*, 1919, **28**, 421.
(b) Demonstration of reconstruction section of defects of hearing and speech. *N. Y. med. J.*, 1920, **111**, 763. *Also* in *Laryngoscope*, 1920, **30**, 487.
22. Sokolowsky. *Beitrage Passow-Schaefer*, 12, 1919.*
23. Skvortzoff. *Sovet. vrach. gaz.*, 1932.*
24. Staton, D. E. Dysphonia and aphonia. *Miss. Doctor*, 1940, **17**, 427-431.
25. Voorhees, I. W. Non-surgical treatment of aphonia (hoarseness). *N. Y. State J. Med.*, 1934, **34**, 53-55.
26. Watson, W. R. Aphonia; report of three cases. *Arch. Otol.*, 1926, **4**, 46-50.
27. Winkelman, N. W. Diagnosis and treatment of hysterical aphonia and diagnosis of malingering in these cases. *M. Clin. North Amer.*, 1937, **21**, 1211-1220.
28. Winslow. *J. Amer. med. Ass.*, 1928.*

*See footnote 1, *supra*.

APHASIA FROM THE VIEWPOINT OF A SPEECH PATHOLOGIST[1]

JEANETTE O. ANDERSON, Ph.D.
Purdue University

I.

This study of eighteen aphasic cases, three with autopsy findings, was made in an effort to synthesize the available knowledge concerning aphasia for the field of speech pathology. From detailed study of the clinical and linguistic manifestations of these patients, an attempt was made (1) to correlate the work that has been accomplished in the field of aphasia with the specialized interests and needs of the speech pathologist; (2) to suggest a concept of aphasia from the point of view of speech pathology; (3) to indicate testing and therapeutic techniques helpful to the speech pathologist in outlining rehabilitory measures for aphasic patients. This article is limited to a discussion of the second of these aims.

Permission was applied for and granted to work with aphasic patients at the State of Wisconsin General Hospital, University of Wisconsin, Madison. Without the whole-hearted cooperation of the chairman of the neuropsychiatric service, Dr. H. H. Reese, and his associates, Drs. Joseph Pessin, Mabel G. Masten and Fritz Kant, this study could not have been made. Residents on the service and members of the nursing staff were also very helpful at all times. Dr. John McCarter of the Department of Pathology graciously made available not only the opportunity and facilities for studying brain sections but also copies of slides and photographs illustrating the pathology found in the autopsied cases. Dr. Robert West, professor of speech pathology and director of the University of Wisconsin Speech Clinic, was more than generous with his time and suggestions in many discussions of the material presented here.

II.

"We learn from history only that we learn nothing from history." This comment is attributed to a contemporary historian. At first reading of a group of case histories of aphasics, a temptation to paraphrase that remark is strong. Upon rereading, however, there appear certain signposts pointing toward the formation of a working concept of aphasia from the point of view of speech pathology. The speech pathologist is not primarily interested in the destruction of brain tissue or the anatomical concept; neither is he concerned basically with the psychological or physiological theories of aphasia *per se;* he is, necessarily, fundamentally interested in the discovery of any master key with which scholars may hope to solve the enigma of aphasia and its concomitant problems.

[1]From a doctoral dissertation, University of Wisconsin, May, 1942.

Although Head and other scholars at various times have attempted to substitute the term *dysphasia* for *aphasia*, they have been unsuccessful; Huhlings Jackson was probably right when he said that in his time the term was in too general usage to be changed (9, 73). Notwithstanding the fact that there is no universal accord in matters of localization at the present time, modern seekers after the truths of the central nervous system and its functioning have learned from history and have come a long way from the doctrine presented by Dr. George Cheyne who wrote in 1734: "The Intelligent Principle, or Soul, resides somewhere in the Brain, where all the Nerves, or Instruments of Sensation terminate, like a Musician in a finely fram'd and well-tun'd Organ-Case; these Nerves are like Keys, which being struck on or touch'd, convey the Sound and Harmony to this sentient Principle, or Musician. . . . The Intelligent principle is like a Bell in a Steeple, to which there are an infinite Number of Hammers all around it, with Ropes of all Lengths terminating or touching at Every Point of the Surface of the Trunk or Case, one of whose Extremities being pull'd or touch'd by any Body whatsoever, conveys a measur'd and proportion'd Impulse or Stroke to the Bell, which gives the proper Sound" (5).

Now, in any anatomical consideration of aphasias, there is a two-directional pull: on the one side toward specific localization of function, and on the other, toward little or no localization. There is almost universal agreement that in right-handed individuals the left cerebral hemisphere is dominant for linguistic functions; atypical cases have been explained on the basis of unrecognized left-handedness, uncrossed pyramidal fibers as a congenital anomaly, or an unrecognized lesion of the left cerebrum (3; 7, 51).

Orton postulates a language area in the left hemisphere and indicates within it areas of localization or of concentration: one for speech and hearing, one for reading, one for writing (4). Most speech pathologists will go along with Dr. Orton and perhaps a little beyond him (19, 16-21). It seems to be fairly generally agreed that within the general language area of the left cerebral hemisphere, there are several areas concerned more especially with one linguistic function than with any others. These areas include:

1. The triangular and opercular portions of the inferior frontal gyrus or Broca's center for the coordinated movements required in speaking (15, 287).
2. The auditory receptive center in the anterior transverse temporal gyrus which lies buried in the floor of the lateral fissure; the area comes to the surface near the middle of the dorsal border of the superior temporal gyrus; the posterior part of the superior temporal gyrus for the comprehension of the spoken word (15, 285, 287).
3. The angular gyrus for the understanding of printed or written language (15, 287).

4. An association region for the comprehension of spoken language in the supramarginal gyrus.
5. Exner's center in the second frontal convolution for the coordinated movements required in writing (9, 47).

The somasthetic region of the anterior border of each parietal lobe and the primary motor region on the posterior border of each frontal lobe play their roles in speech by making possible the feeling and actuating of the movements of the muscles used in speech (19, 19).

Although the speech pathologist does recognize these several areas and their more than general concern with certain aspects of the comprehension and production of language, he is fully cognizant of the tendency of the entire brain to function as a whole in all linguistic activities (7, 50). If any one area is affected adversely, the entire mechanism is disorganized and there is dysfunction in activities other than that mediated directly by the damaged area. Crippling in any part lames the whole neurological apparatus for language.

In order to establish some point of departure, a brief discussion of a comprehensive contemporary concept of aphasia seems apropos. The concept of aphasia, apraxia and agnosia held by Nielsen is perhaps the most comprehensive and critical evaluation of the published material in this field. Nielsen in his writings takes into account all the work published in the name of aphasia from pre-Broca conjectures to the Weisenburg-McBride investigation and subsequent studies. He commences with the premise that: "Language is a highly complex cerebral function requiring for its use the entire brain" (9, 17). He goes on to say, "It is impossible for any single approach to the problem of aphasia to be successful. All three methods of approach are simultaneously to be considered: psychological, physiological and anatomical" (9, 19). Nielsen makes very clear the fact that aphasia is neither motor nor sensory in character but is fundamentally associational. The aphasic disturbance, however, may seem to be more sensory or more motor in character, depending upon the location of the causative lesion. If aphasia is to be considered as it appears clinically, it must be studied correlately with agnosia and apraxia. When agnosias and apraxias affect the use of linguistic symbols, they become forms of aphasia, according to Nielsen (9, 30-31). Certain distinctions should be recognized and observed in the usage of the three terms: *agnosia, apraxia, aphasia.*

According to Nielsen, *agnosia*, the term proposed by Freud, signifies loss of ability to recognize objects or symbols "through one sense organ, the loss being due to a cerebral lesion, and general mental defect excluded as cause, relative intactness of the sense in question retained, and the object or symbol being recognizable through some other sense organ. It is literally possible for two simultaneous agnosias to be present, but in all cases the loss must be in primary identification" (9, 165).

Apraxia, discussed by Nielsen in accord with modern thought stemming from Liepmann's study in 1900, "is considered a motor disturbance due to involvement of the association fibers of the cerebrum (9, 33) . . . . resulting in the inability of the patient to apply his powers to a purpose, the disability not depending on paralysis, incoordination or dementia" (9, 190). It may be added that disability in apraxia means disability for activities with which the patient has been familiar; he can no longer perform functions to which he has been accustomed.

It may be recalled that Nielsen's definition of aphasia takes into consideration the agnosic and apraxic bases of many aphasic disturbances: "Aphasia is the term in common use for all disturbances of language, motor or sensory, due to a lesion of the brain but not due to faulty innervation of the musculature necessary for speech or to involvement of the sense organs themselves or to general mental defect. Aphasia includes all agnosias and apraxias which have to do with language" (10, 265).

Nielsen's concept of aphasia, then, seems to be this: all disturbances of symbolization are basically agnosic or apraxic in character. Aphasia is, however, impairment on a higher functional level than either recognition or performance; it includes defects in linguistic symbolization and significance of language and may be caused by lesions affecting association fibers and regions. Agraphia and alexia are both considered aphasic manifestations, but Nielsen cautions that they should be diagnosed differently in terms of causation, i.e., agnosia, aphasia, apraxia. Furthermore, two divisions are recognized in aphasia: aphasias on the lower levels and aphasias on the higher levels. The first group includes expressive and receptive disturbances so severe that they are easily recognized; the second applies to cases in which language is limited quantitatively rather than qualitatively.

This concept of aphasia is a cornerstone for the speech pathologist in the building of his concept. Nielsen is interested primarily in the localizing value of aphasic manifestations and says so. The speech pathologst is concerned with this aspect of aphasia only incidentally; his basic problem is to determine the type and extent of impairment and the residual language potential, and to translate his findings into prognosis and therapy for speech. In order to realize these objectives, he must, it is true, have a general knowledge of the cerebral mechanism for language and of cortical regions of localization for linguistic functions. He need not be concerned with exact localization.

If there is damage in the areas for visual recognition and comprehension of language symbols, the resulting aphasia manifests itself in disorders of reading and sometimes of writ-

ing; it is fairly generally agreed that subcortical destruction in the region of the angular gyrus causes alexia and that cortical damage to this gyrus produces agraphia as well (10, 236). Sometimes the impairment is on the recognitive level; if it is, the patient can read no symbols at all. If the disorder is on the comprehensive level, the patient can recognize symbols and sometimes read words; unless the words are read aloud, however, they mean nothing to these patients. When impulses enter the brain on an auditory level rather than a visual one, the proper associations can be made and the linguistic symbols become meaningful. This finding suggests that visual compre-

FIGURE I. Frontal section through the temporo-parieto-occipital region of the brain of H.P., showing the tumor mass and hemorrhagic infarcation that destroyed parts of the temporal and occipital cortices in the left hemisphere. This patient retained ability to write but not to read or to revisualize. He exhibited visual receptive aphasia and finally agnosia; alexia; anomia; impairment of higher functions. (Photograph by staff photographer, State of Wisconsin General Hospital.)

hension is impaired and that auditory understanding is relatively intact. It is believed by a number of investigators that there is a writing center in the second frontal convolution (Exner's writing center); it is suggested that this is the motor formulative writing area, as Broca's region is believed to be an area for motor coordination for speech (11). The cortex of the angular gyrus is believed to mediate comprehension and integrated conception of the symbols needed for the formulation of written speech; the region in the second frontal convolution is thought to formulate the motor production of these symbols (9, 49). In any event, it has been demonstrated that a lesion in either area can cause agraphia. Alexia may be caused by a subcortical lesion in the angular gyrus region or in the vicinity of Broca's center for motor speech. In one instance the agraphia or alexia is on a primarily receptive basis; in the other, it is predominantly expressive in character. (See Figure 1.)

In cases of damage in the region of the subcortical angular gyrus, agnosia or aphasia may occur not only for symbols but also for objects and colors. In these cases, the resulting inability to produce names at will is called anomia; the areas for revisualization seem unimpaired, and the patient can often describe the object and its use, sometimes give synonyms for the required word and occasionally even name objects if they are described and not presented. There is frequently a concomitant homonymous hemianopsia or blindness for one-half the visual field in these cases. This is true because damage to the mesial occipital cortex of the dominant occipital lobe causes blindness in both eyes for the opposite lateral half of the field of vision. The major hemisphere seems to dominate in recognition and comprehension above the perceptive level; if the dominant occipital area is damaged, the inferior lobe will continue to function on a visual basis but will tend to be powerless in the higher functions of visual recognition and comprehension. It is as though the lens of a camera were opened and there was no film upon which the admitted light could fall—there is nothing to develop.

Damage to the superior convolution of the left temporal lobe can cause partial deafness or auditory aphasia, or both, depending on the location of the lesion. If the damage is in Heschl's convolution, perception deafness in the contralateral ear results;[2] if the lesion is in the posterior part of the superior temporal gyrus, auditory aphasia and all its concomitants may ensue. When hearing is intact on the recognitive level but impaired for comprehension of language sounds as language, it is as if a phonograph record had been cut. The record has everything speech has, but has not yet been returned or transmuted into sound waves; it is a record of all the oral language activity that has taken place, but it is not speech until it has been played back or retranslated into sound waves.

It has been demonstrated several times that surgical or pathological removal of the dominant temporal lobe results in partial deafness and in auditory aphasia (**14, 8**). When the aphasic manifestations are lessened in such cases, it is assumed that the minor temporal lobe takes over the functions of linguistic recognition and comprehension usually performed by the major. The minor lobe usually starts by taking over for only a few minutes at a time; although this time is gradually increased, the non-dominant lobe does not tend to acquire the powers of concentration or of assimilation that had been built up by the dominant lobe (**12**).

[2]Total deafness of either ear from damage to the auditory pathway within the brain is rare because fibers from both right and left cochlear nuclei are probably included in the lateral lemniscus of each side of the brain.

In these cases the patient usually is able to recognize and to comprehend linguistic symbols visually; he seems to have no trouble in formulating and producing language, but his speech tends not to be meaningful because he has no auditory control; he perceives sound, but not symbolic sound. Thus he suffers not from auditory agnosia but from auditory aphasia.

In cases where both visual and auditory aphasias are present, the resulting syndrome is that described by Wernicke and is known as Wernicke's aphasia, or as sensory aphasia. It is important for the speech pathologist to remember that sensory or receptive aphasia may be either auditory or visual, or a combination of the two.

There may be a third basis for receptive aphasia. If there is any reduction of sensitivity or blocking in afferent fibers carrying tactile or kinesthetic impulses, there might be failure of association between such fibers and fibers mediating motor response. Spoken language is formulated from auditory, kinesthetic and in a smaller part from visual and cutaneous images (**19, 19**). Visual and auditory agnosias and their place in the aphasic problem have been considered in some detail. It seems reasonable that a cutaneous or kinesthetic agnosia could lead to similar disturbances of association; such dysfunction would tend to produce receptive or sensory aphasia on a tactile or kinesthetic basis. When children learn to read, they say the words aloud at first, later the words are whispered, and finally, as the child develops habits of silent reading, the motor response becomes entirely implicit or at least undiscernible to others. Reading, however, is probably aided by not only visual recognition and interpretation of symbols but also by kinesthetic imagery, if not by actual subvocal motor response. Alexia, then, could occur on one of two receptive levels: visual or tactile-kinesthetic.

Any predominantly receptive aphasic disturbance produces aberrations in spoken language. It has been mentioned that visual aphasias may be the focal cause of inabilities in naming and reading because of imperfections in the recognition and comprehension of objects and symbols; when these are not recognized visually or when they have lost their symbolic significance, they cannot be translated by way of association areas into the motor formulations and productions we call written or spoken language. In like manner, when both internal and external speech has lost its symbolic significance and is perceived acoustically only as unfamiliar speech, there is double reason for dysfunction in the production of spoken language symbols. In the first place, there is no guide for correctness of sound; in the second, there is little motivation to use speech in response to environmental stimuli; control of language by language is no longer possible, either implicitly or overtly.

Man is accustomed to produce linguistic symbols, both written and spoken, with the aid of as many devices as he can command. Auditory, visual, tactile and kinesthetic imagery are called into use in the production of speech. When one of these media is impaired, each of the others suffers.

For a patient who had been predominantly "eye-minded" or who had relied most extensively upon visual imagery as a guide to accurate production and correct pronunciation of spoken language symbols, an auditory aphasia would cause less serious phonetic lapses in speech than a similar affection in an "ear-minded" individual. If the patient had been accustomed to "hearing" the word instead of "seeing" it before speaking it, if his imagery were primarily auditory, an auditory aphasia would have serious effects upon his spoken language in all probability. It is not difficult to imagine how a similar chain might operate in the production of written speech; for example, a patient used to "seeing" the words as he wrote them probably would be agraphic if he suffered a visual aphasia. A pursuance of this discussion leads directly toward a consideration of predominantly expressive disorders. When overt speech cannot be produced because of the patient's inability to formulate kinesthetically and synergically the conventional phonetic combinations that his group calls speech, he is said to have an expressive or motor aphasia. If there is no accompanying dysarthria, if the motor area and tracts are intact, the defect is believed quite generally to be in the posterior region of the major (usually left in right-handed individuals) hemisphere. This type of aphasia varies somewhat in its manifestations with the location of the lesion causing it.

The causative lesion of cortical motor or Broca's aphasia has been localized in the cortex of the left third frontal convolution, but it may extend into neighboring parts of the frontal lobe and even into the insula (18, 58-60). The patient suffers grave defects of both speech and writing, although he may be able to say and to write his name and a few other very familiar symbols. This is the "verbal aphasia" of Head and the "central aphasia" of Goldstein (18, 58-60). (See Figure 2.)

Subcortical aphasia of the motor type supposedly affects speech but not writing; few cases have been reported in which this situation obtained. The causative lesion in this type of affection used to be thought of as subcortical in the region of the third frontal convolution; now, however, there is little agreement concerning the location of the pathology in this aphasic picture (18, 56-58).

In transcortical motor aphasia the patient is usually able to repeat speech and to write from dictation and to copy, although he cannot speak or write spontaneously. There is much disagreement concerning the localization of the causative

lesion; it has been suggested that a partial lesion of the cortex in the region of the posterior part of the third frontal convolution may be responsible for this type of motor aphasia (9, 76-77).

Careful case reports indicate that these so-called types of motor aphasia are not as clear-cut as they were once thought to be, and that there are frequently complicating manifestations and overlapping manifestations between and among them. In almost every instance, the expressive inabilities are accompanied by greater or lesser general intellectual losses.

FIGURE 2. Frontal section through the brain of E.O., showing abscess under the superior part of the left motor area and edema of the left cerebral hemisphere. This patient exhibited expressive aphasia for spoken and perhaps for written language. (Photograph by staff photographer of State of Wisconsin General Hospital.)

Still another expressive or motor aphasic manifestation may be mentioned. Alexia has already been discussed as a result of impaired visual or tactile-kinesthetic reception. Reading, however, is a function of eye-movements as well as of visual recognition and interpretation. Two bases are postulated for word-blindness. One is a lesion in the region of the angular gyrus; the other is a deficit in psycho-visual orientation. This second dysfunction is also known as geometric-optic agnosia, or loss of sense of direction of lines, and is a part of disorientation in space; it is the aphasic manifestation of the larger problem. It is manifested not in failure of recognition but in inability to reproduce or to follow lines, figures or symbols with motor activity of the eyes or hands. Pötzl and Henschen have each reported cases, and Pötzl suggests that the occipital convex cortex is chiefly concerned (9, 60). This does not explain completely the ability of some patients to recognize objects and symbols, but to experience difficulty in left-to-right spatial orientation in reading. In such cases, the loss would seem to be a motor one unless it can be traced directly to a lack or disorder of cerebral dominance. The loss may be a partly motor one, also, when the impairment seems to be kinesthetic; kinesthetic

images are built through and help to establish movements of the speech musculature. Thus it seems quite possible that alexia may be either sensory or motor in character.

Any primarily expressive disorder reduces to greater or lesser extent the functioning of the entire cerebral mechanism for language. Most people retain some vestiges of the childhood habit of saying or whispering a word as it is written. In cases of predominantly expressive aphasia, writing is probably impaired in almost direct proportion to the degree of retention of this habit; if the tendency toward kinesthetic imagery is strong, the impairment of such imaging will, of course, be apparent in all activities in which it has played a motivating or stimulating role. Moreover, if strong kinesthetic imagery had been used as a prop in visual and auditory comprehension, any dysfunction in such imagery would bring about corollary visual and auditory dysfunction. Thus it becomes apparent that the snapping or weakening of any arc in this most complex of all neurological chains almost inevitably causes more or less marked disturbance throughout the system.

Since it is true that no one part of this linguistic mechanism functions unto itself alone, it is important in testing aphasic patients to determine why there is no overt speech before diagnosing the disturbance as primarily expressive. Sometimes the patient cannot formulate and produce the words although he knows exactly what he wants to say, may say it implicitly and recognizes the words he wants when he hears them. In other cases, however, the patient is speechless because he has nothing to say. According to Abbott **(1)**, the patient suffering from Broca's aphasia is speechless but not wordless; he maintains that a parietal lesion seems to cause loss of ideation as well as of word-formulation. Raney **(13)**, however, reports two cases in which surgery in the left parietal area caused no permanent aphasia. There are indications that in some patients parietal lesions cause varying clinical pictures and aphasic manifestations. If the patient not only cannot speak, but also gives no indication of knowing what he wants to say, the lesion is likely to be in the dominant parietal lobe **(6, 2)**. Amnesic aphasia may be caused by a parietal lesion, although this form of aphasia has not been localized definitely. Unless testing is adequate, this aphasic manifestation may be mistaken for or confused with expressive aphasia; amnesic aphasia is basically receptive.

An aphasia for overt speech thus may be on a primarily receptive or expressive basis, depending on the location of the causative lesion. Aphasic manifestations are not always as they seem at first cursory examination. Even in apparently circumscribed lesions, damage to one specific brain area almost certainly would cause repercussions in other language activities. When the causative lesion is vascular or neoplastic, as it is so

frequently in cases of aphasia (eighty-three per cent of the sixty cases described in the Weisenburg-McBride study; sixty-one per cent of the cases in this investigation), there is anatomical as well as physiological and psychological basis for the impurity of the aphasia and its observable manifestations. (See Figure 3.) The posterior part of the third frontal convolution,

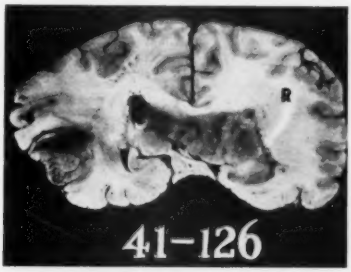

FIGURE 3. Frontal section of the brain of R.P., showing destruction of structures by two large tumor masses. This patient exhibited visual and auditory receptive aphasia and probably agnosia; expressive aphasia for spoken and written language; ideational apraxia. (Photograph by staff photographer, State of Wisconsin General Hospital.)

the frontal lobe writing center, the internal capsule, the superior temporal convolution and the temporo-parieto-occipital region are all supplied by the middle cerebral artery. A lesion in the main trunk of this vessel would affect all of these areas; a lesion in any one of its branches would tend to have temporary or permanent effects upon the blood supply to at least one of these areas directly and to the others indirectly. A neoplasm in the Sylvian or insular region or in the frontal, temporal, or parietal lobes would exert pressure on these language areas; an occipital lobe neoplasm would affect at least the posterior region of the language zone. Therefore, in most cases of aphasia, there is not only functional but pathological interruption of normal activities of both reception and expression; the tendency is toward mixed and away from pure aphasias.

Those investigators who have recognized most clearly the impurity and complexity of the problem in aphasia have attempted to find solutions to them through a primarily psychological approach. Jackson's position was that aphasias are disorders of propositionizing; Head suggested that aphasias are all disturbances of symbolic formulation; Weisenburg and McBride pointed out the fact that aphasic disorders are rarely clearly defined. These students have been interested in the behavior of the brain as it comprehends and produces language. Although the speech pathologist, too, is concerned with the

receptive and expressive formulation of linguistic symbols, he is even more interested in the restitution of function than in gross or specific losses or even remnants of linguistic ability. Granted that he must study function, insofar as he or anyone may, before he can try to re-establish it, his reach for understanding must extend beyond that of the anatomist, physiologist or even the psychologist. It is not enough for him to realize the extent of the damage, to have an idea of functioning in both physiological and psychological terms; he must plan to use the uninjured neural tissue and to reroute language activities over, to and through this sound brain-stuff.

From the point of view of speech pathology, aphasia includes all linguistic disturbances, expressive or receptive, caused by a lesion of the brain but not attributable to faulty innervation of the musculature used in speech or writing, nor to pathology of the sense organs themselves, nor to general mental deficiency; these language disturbances are to be understood as failures in the symbolization, retention and production of mental concepts in association with conventional phonetic and cheirographic symbols. The speech pathologist is interested in the specific language losses that make up or cause the usual mixed aphasia. He is concerned with a classification of these disturbances that will indicate the underlying anatomical, physiological and psychological bases of the dysfunction; this means, of course, that his knowledge of the interrelationships of these fields within the aphasic frame must be so familiar to him that he can, from the point of view of any single specialist, see the picture in three dimensions. From the point of view of one who must make use of the undamaged brain areas for restitution of function, he will add his own analysis of the disorder in terms of speech prognosis and therapy.[1] Thus the speech pathologist's concept of aphasia should be not only as precise as that used in localization by the surgeon; it must be as general as that of the psychologist who insists that the linguistic mechanism is a *Gestalt* and functions wholly. The speech pathologist must, more than any other investigator in this field, see both the parts and the whole clearly and simultaneously in their mutual relationships. It is not enough for the speech pathologist to see clearly and in proper perspective aphasias and their various concomitants and combinations; he needs to evaluate linguistic failures in terms of agnosia and apraxia and to be able to recognize them as corollaries of apraxia and agnosia manifested in non-language activities. If he is to assume his responsibilities and discharge his duties, the speech pathologist needs a revised classification of aphasias.[4]

Weisenburg and McBride (18) in 1935 suggested the most useful basic descriptive classification of aphasias. However, the difficulties encountered in classifying patients in their own

study and in subsequent studies by other investigators suggest that this classification leaves something to be desired. It has been suggested that the terms *expressive, receptive* and *amnesic* are general descriptive words that do not indicate either the impairment of function or the area of damage. In attempting to clarify this state of affairs, especially in the interests of the speech pathologist, there are several matters to be considered. Any classification we adopt should be consistent, specific, simple and not too cumbersome for general usage.

Although Nielsen is concerned primarily with the localizing value of aphasic manifestations, he does recognize the possibility and worth of speech therapy (**10**, 258, 277-278). For this reason, perhaps, his classification of aphasias is more useful to the speech pathologist than are the others in the literature. However, even this most recent categorization does not quite serve the speech purpose. In this connection, it seems significant that no classification has been generally accepted; moreover, there is no indication that one will be.

Nielsen's classificatory system is quite specific and is relatively simple. It does, however, make any form of expressive aphasia apraxic in theory. Unless we subscribe to the outmoded Liepmann doctrine of "motor" aphasia as apraxia, we are almost certain to be disturbed by this characterization of the disorder. Although Nielsen in his discussion of cases recognizes that aphasia and apraxia should be differentiated, his classification leads one to believe that he considers "motor" or expressive aphasias forms of apraxia. This may be partly true, but it does not tell the whole story. As Nielsen noted, there are expressive aphasias for speech, for writing and for both; these may be considered as linguistic apraxias under the limb-kinetic designation of Liepmann. Total aphasias in which the patient cannot speak because he has no ideation could be classified with the ideational or even with the ideo-kinetic apraxias described by Liepmann (**18**, 104-106).

In transcortical aphasia, however, it is not the motor side of language that is impaired; when the patient is asked to repeat words and even sentences, he can do so. He does this upon request and wills to do it. There is impairment of cortical stimulation for speech; but when the impulse comes from Wernicke's region and goes directly to the region for motor speech without need for any functioning on a conceptual level, there is no apraxia for speech, written or oral. There is no agreement as to the location of the lesion in this clinical picture (**18**, 61-62).

In the expressive aphasia usually seen clinically, there is apparently apraxia for words as concepts and not for words as phonetic combinations. The patient can and does produce all

sounds in isolation or combination at one time or another and seems to have no more trouble with the production of the difficult [s], [ʃ], [θ], [ð], [r] and [l] than with the relatively easy vowel and [p], [b] and [m] sounds. Therefore, these aphasic individuals have no apraxia for linguistic symbols but do have what might be called an apraxia for linguistic concepts. Apraxia, properly, means inability to handle concrete objects. When the inability is in the realm of the intangible, as it is in the case of linguistic concepts, the disorder is not apraxia, but aphasia. Furthermore, these patients use the speech musculature in other activities; they "apply their powers to a purpose" in eating, swallowing, breathing and sometimes even in singing (17, 728). Thus the disturbance is not an apraxia, according to Nielsen's definition, and should not, in the interests of clarity, be classified as apraxia. When the muscles used in speaking and writing can perform other voluntary functions successfully, the disorder is peculiar to linguistic activities and should be called an aphasia.

In connection with this differential diagnosis, there is one phenomenon that does not fit clearly into either the aphasic or the apraxic category. In this study six patients with predominantly expressive aphasias or with mixed aphasias, including expressive manifestations, had marked glosso-apraxia for protrusion of the tongue. These patients could wrinkle their foreheads or noses, purse and retract their lips, puff out their cheeks, but they could not protrude their tongues on oral or written request. Sometimes they could point to their tongues and move them in imitation of the examiner; sometimes they could not. Immediately after their failures, however, all six of these patients would at times lick their lips or use their tongues in ways not suggested to them linguistically. This apraxia peculiar to the tongue cannot be explained on the basis of the probable completely crossed upper motor neurones of the twelfth cranial nerve, because the same pattern of crossing obtains for the branch of the seventh cranial nerve innervating the face below the eyes, and the lips and cheeks of these patients can be pointed out and moved on request. Although similar observations have been reported from time to time in the literature, no adequate explanation has been offered for this lingua-phenomenon. These patients exhibit a real glosso-apraxia but tend to show no apraxia of other musculature except for linguistic functions.[3]

These findings indicate that loss of expressive language abilities cannot be explained satisfactorily in terms of apraxia; there is more than failure of motor formulation involved; there

[3]This lingual disturbance makes the term "linguistic" seem especially applicable to these aphasic disorders.

is failure of motor formulation for language. Dr. Nielsen's inclusion of motor aphasias as apraxias in his classification and his recognition of aphasic disturbances as more than apraxia tends to be confusing at times. His concept, discussion and definitions are clearer than his classification; a familiarity with and understanding of them is requisite for an appreciation of his classificatory system.

III.

The speech pathologist can follow the psychologist in his concept of aphasia as a disorder of propositionizing or of symbolic formulation; he can agree with the physiologist that aphasia is caused by an interruption of association pathways and areas; he can say with the anatomist that certain cerebral areas tend to subserve specific language functions. Moreover, he can postulate with Nielsen that agnosias and apraxias belong in the aphasic picture and that psychological, physiological and anatomical aspects of the problem ought to be considered in parallel.

If he is to be free to study and to make his own contributions, however, there are some traditional tenets to which the speech pathologist can give little credence. He cannot agree with many psychologists that there is little or no localization of function; neither can he subscribe to exact anatomical localization. In general, he can follow the lead of the physiologist in admitting that certain areas subserve certain linguistic functions more than others and that the entire brain is needed for normal linguistic activity. In order to discover aphasic manifestations and to prescribe techniques of special re-education in speech, the speech pathologist needs to consider not only three aspects of the problem, but a fourth as well: the possibility of retraining in terms of all three. He needs, first of all, a consistent classification of aphasic manifestations. This classification should be based upon manifestations actually observed, should be exhaustive, and should recognize aphasia as the basic linguistic disturbance of which some agnosias and some apraxias are parts. Aphasia is greater than the sum of its parts. In order to minimize confusion and to serve maximally, this classification should be couched in simple terms capable of only one interpretation in any field of study.

The following classification of aphasic manifestations is suggested as a first step toward a practical solution of the problem:

I. Expressive manifestations
   A. Implicit (probably caused by a parietal lesion)
      1. Ideational aphasia
      2. Ideo-kinetic aphasia
   B. Overt (probably caused by a frontal lobe lesion)
      1. Spoken language

a. Broca's aphasia (third left frontal convolution, cortex)
b. Subcortical aphasia (subcortical Broca's region)
c. Transcortical aphasia (questionable)
2. Written language
a. Agraphia (Exner's writing center, second frontal convolution)

II. Receptive manifestations
A. Auditory (superior temporal convolution)
1. Agnosia (Heschl's convolution)
2. Aphasia (posterior part of superior temporal convolution)
B. Visual (temporo-parieto-occipital area)
1. Agnosia
a. For linguistic symbols
b. For objects and colors
c. Geometric-optic
2. Aphasia
a. Alexia
b. Agraphia
c. Anomia
C. Other sensory media
1. Tactile
2. Cutaneous
3. Kinesthetic

III. Associative manifestations
A. Aphasia on lower levels
B. Aphasia on higher levels

IV. Combinations of manifestations
A. Wernicke's aphasia
B. Total aphasia
C. Other combinations of aphasic manifestations

V. Manifestations indicating special abilities useful in speech therapy
A. Expressive
1. Oral facility for building new motor synergies
2. Manual facility for building new motor synergies
3. Kinesthetic imagery
B. Receptive
1. Visual imagery
2. Auditory
a. Imagery
b. Memory span
3. Other sensory imagery
C. Associational
D. The X-factor (16)

This suggested classification of manifestations includes the generally accepted aphasic duality: expressive and receptive disorders. Moreover, provision is made for general dysfunction and for the recognition of specific impairments within the general one. Although its fundamental purpose is to guide the speech pathologist in diagnostic, prognostic and therapeutic procedures, this classification may prove of some general use in the examination of aphasic patients by other specialists.

On the basis of this study of eighteen aphasic patients, no attempt is made to classify types of aphasia. There are certain aphasic manifestations that can be grouped under the several headings; a given patient may exhibit almost any combination of these signs. If he shows more manifestations from one group than from any other, he may be described as suffering

from, for example, predominantly expressive aphasia. In most cases of aphasia, however, it seems more satisfactory to diagnose the case as aphasia and then to list the linguistic findings. In no other way can an adequate picture of the patient's losses, psychological, physiological and anatomical, be seen. The very confusion within the field of study for over eighty years suggests that the attempt to classify aphasic types is not only unsatisfactory but futile. This may be because there are no aphasic types. This study, although based upon relatively few cases, indicates that there is one fundamental linguistic disturbance: aphasia; that this disorder has many clinical manifestations that tend not to appear singly but that may be exhibited in almost any combination.

On the basis of these studied cases of aphasia, the speech pathologist seems justified in postulating a synthesized concept of aphasia. He need not quarrel with existing definitions of aphasia. He must insist that classification of aphasic cases is arbitrary and not natural; confusion resulting from such classification may be minimized by classifying manifestations and not cases. This suggested classification is based upon clinical observation and not upon traditional theory; it allows, however, for all substantiated observations of aphasic signs and symptoms.

A basic concept of aphasia for the speech pathologist evolves: aphasia is a linguistic impairment; there tend not to be "pure" aphasias; each aphasic patient exhibits a disturbance made up of certain aphasic manifestations peculiar to the given case; any classification is of aphasic manifestations and not of aphasias. Examination reveals the manifestations; they form the specific aphasic picture seen in each case. Once he has this concept of the inconstancy, unpredictability and irresolution of the aphasic problem, the trained speech pathologist is ready to examine and to attempt re-education of aphasic patients.

IV.

1. There seems to be no universally satisfactory classifition of aphasias.
2. A study of aphasia made from any one specific point of view is incomplete.
3. There appears to be a general language zone in the brain within which several specialized areas for more or less specific linguistic functions may be designated.
4. A concept of aphasia as a basic disorder that may exhibit any combination of many aphasic manifestations seems useful for the speech pathologist. These manifestations may be classified as expressive, receptive, associative, mixed.

REFERENCES

1. Abbott, Kenneth H. Total aphasia from a parietal lesion. *Bull. Los Angeles neurol. Soc.*, 1939, 4, 84-86.

2. Austregesilo, A. Aphasie et lobe pariétal gauche. *Pr. Méd.*, 1940, **48**, 126-132.
3. Bryngelson, Bryng. Investigations in the etiology and nature of dysphemia and its symptom, stuttering. *J. Speech Disorders*, 1942, **7**, 15-27.
4. Chesher, E. C. Aphasia, technic of clinical examinations. *Bull. neurol. Inst. N. Y.*, 1937, **6**, 134-144.
5. Cheyne, George. The English malady: or a treatise of nervous diseases of all kinds. 1734. (Cited by Ingham, Samuel D. Consciousness in relation to the cerebral localization. *Bull. Los Angeles neurol. Soc.*, 1937, **2**, 147-155.)
6. Courville, C. B. Otogenous abscess of the parietal lobe. *Bull. Los Angeles neurol. Soc.*, 1936, **1**, 65-69.
7. McKendree, Charles A. Neurological examination. Philadelphia: Saunders, 1928.
8. Morel, Ferdinand. L'audition dans l'aphasie sensorielle. *Encéphale*, 1935, **30**, 533-553.
9. Nielsen, J. M. Agnosia, apraxia, aphasia. Los Angeles: Los Angeles Neurol. Soc., 1936.
10. ————. A textbook of clinical neurology. New York: Harper, 1941.
11. ————. The unsolved problems in aphasia. I. Alexia in "motor" aphasia. *Bull. Los Angeles neurol. Soc.*, 1939, **4**, 114-122.
12. Nielsen, J. M., and Raney, R. B. Recovery from aphasia studied in cases of lobectomy. *Arch. Neurol. Psychiat.*, 1939, **42**, 189-200.
13. Raney, R. B. Operation in left parietal region without total aphasia. *Bull. Los Angeles neurol. Soc.*, 1936, **1**, 17-19.
14. Raney, R. B., and Nielson, J. M. Complete major (left) temporal lobectomy. Abstract of psychic changes. *Bull. Los Angeles neurol. Soc.*, 1936, **1**, 69-72.
15. Ranson, Stephen W. The anatomy of the nervous system. (6th ed.) Philadelphia: Saunders, 1941.
16. Twitmeyer, E. B., and Nathanson, Y. S. Correction of defective speech. Philadelphia: Blakiston's, 1932.
17. Ustvedt, Hans Jacob. Ueber die Untersuchung der Musikalischen Funktionen bei Patienten mit Gehirnleiden, besonders bei Patienten mit Aphasie. Helsingfors: 1937.
18. Weisenburg, Theodore, and McBride, Katharine E. Aphasia: A clinical and psychological study. New York: Commonwealth Fund, 1935.
19. West, Robert, Kennedy, Lou, and Carr, Anna. The rehabilitation of speech. New York: Harper, 1937.

A PHONETIC APPROACH TO THE PROBLEM OF PERCEPTION IN A CASE OF WERNICKE'S APHASIA

MARY HUBER, M.A.
Brooklyn College

I. INTRODUCTION

Wernicke's aphasia is recognized as a syndrome characterized by loss of comprehension of spoken words (acoustic verbal agnosia), loss of comprehension of written words (visual verbal agnosia) and agraphia (2, 200). Wernicke has assigned the cortical lesion resulting in this syndrome to the posterior half of the first, and adjoining portion of the second, temporal convolution in the dominant hemisphere.

The present study, which is the first of a series of investigations of verbal perception in receptive dysphasia, was inspired by a recent article by Hemphil and Stengel on pure word deafness (1). While these authors were mainly interested in discovering the relationship, if any, between high frequency deafness and pure word deafness, the very comprehensive verbatim records of their patient's paraphasic speech led the present investigator to wonder whether an analysis of the phenomena of paraphasia and perseveration from a phonetic standpoint might not reveal some interesting clues to the linguistic mechanisms of receptive dysphasics. These patients themselves, whose spontaneous speech is occasionally intelligible and often voluble, have, at times, attempted to explain their difficulties in expression and comprehension. Lordat, a Frenchman, who had written a book on speech, became aphasic in 1828 and complained that the speech of others came too fast for him to comprehend it; he used the wrong words, and in trying to correct himself, again spoke unintended words (2, 2). Hemphil and Stengel's (1) patient offered the following vivid description of his difficulties in auditory comprehension:

> It does not finish. Its one on top of the other. The words split themselves up. I hear what you are saying but the pronunciation does not finish. The voices seem to be as clear as they should be. I can hear you quite plainly but all come on top. I can hear you dead plain but I cannot get what you say. The noises are not quite natural. I hear your voice but not the words . . . . It does not pronounce itself.

The phenomenon of auditory verbal paragnosia, seemingly the mis-hearing of words, experienced by receptive dysphasics and the characteristic paraphasia and jargon aphasia have interested the present investigator over a period of several years. The mechanism of perseveration, which is the repetition of a previously uttered word, instead of the intended word, and the extent to which it is responsible for the neologisms manifested in jargon aphasia deserves a more systema-

tic analysis than has thus far been attempted, to the best of our knowledge.

Many angles of the approach to this problem have been considered. It was finally decided to begin with an investigation of the patient's ability to repeat after the examiner a series of verbal symbols classified from the standpoint of making a critical analysis of the difficulties they represent either in perception or initiation of the response.

In selecting appropriate subjects for this testing program it has been necessary to establish some criteria which would help to minimize the possibility of unwittingly including individuals whose dysphasic problems were not predominantly receptive. In a general way it has usually not been difficult to eliminate those dysphasics suspected of motor involvement, because thus far we have insisted on using only such patients whose spontaneous speech was rather profuse as compared to that of the average motor dysphasic whose vocabulary, intelligible or otherwise, is exceedingly limited. The occurrence of hemiplegia, particularly of the spastic type, is, of course, more frequently observed in connection with motor dysphasia than with the sensory type. Many authorities, too, regard Broca's aphasia and subcortical motor aphasia as types of apraxia and a careful examination of the voluntary non-verbal activity of the speech mechanism has been found useful in the differential diagnosis of motor and sensory aphasia. Knowledge as to the location of the cortical lesion is, of course, among the most useful criteria, but it is not always available nor entirely reliable in the living, ever-changing human organism.

We are aware that what we are testing may not always be perception, but quite frequently impaired initiation, since we are considering the results of our tests in the light of the patient's ability to reproduce what he hears. It is hoped that in the course of our investigations we may discover under what conditions failure of perception is responsible for an incorrect response, as compared to difficulties resulting from impaired initiation of a truly dysphasic nature not seriously complicated by motor or dyspraxic involvements.

In the interests of clarity at this point a brief mention of the incidence of echolalia in connection with receptive dysphasia is in order. Subjects classified under the category of transcortical sensory aphasia have manifested the ability to repeat accurately, but uncomprehendingly, anything that they hear. The presence of echolalia has been used as a criterion for excluding these subjects from the present study as not true representatives of Wernicke's aphasia.

II. The Problems

The tests were constructed with the following objectives in mind:

1. To analyze the paraphasia, or jargon aphasia, of the receptive dysphasic from a phonetic standpoint, in order to discover what, if any, relationship exists between the patient's difficulties in perception of specific verbal units and his ability to repeat a stimulus sound or word after the examiner.
2. To observe the relative difficulties in perception and repetition of vowels as compared to consonants.
3. To determine whether the sonority or surdity of a given sound influences the subject's perception and ability to initiate the correct response.
4. To compare the relative difficulty in perception of the test symbols when presented (1) with only the auditory stimulus, and (2) with both the auditory stimulus and the additional cue of lip reading.
5. To discover if the receptive dysphasic experiences less difficulty in perceiving and reproducing isolated acoustic verbal symbols as compared to acoustic verbal symbols combined into words.
6. To observe whether the number of syllables in a test word has any relation to the subject's difficulty in perceiving and reproducing the symbol.
7. To determine whether the rate of presentation of the test symbols influences the extent to which perseveration interferes with initiation of a correct response.
8. To analyze the results of the investigation from the standpoint of providing more effective procedures in the linguistic rehabilitation of receptive dysphasics and possibly dysphasics in general.

III. The Case Report

The subject was a 22-year-old white male. He had completed three years of high school and had been working on his father's farm until a few weeks before his admission to the neuropsychiatric service of the Wisconsin General Hospital. His complaint was a right-sided hemiplegia with aphasia. According to the patient's local physician his condition resulted from a period of illness believed to be pneumonia. An encephalogram revealed no displacement of the ventricular system to indicate the presence of a tumor. In the upright projections there was adequate visualization. The right lateral ventricle was normal in size while the left showed a slight enlargement especially in its anterior and midportion. The septum was in the midline. The third and fourth ventricles were fairly normal. There was good visualization of the basal cisterns and they were of normal size. There was quite good filling of cortical pathways over the right hemisphere with normal channel. On the left, however, there was incomplete filling with only the midline and superior sulci visible, while laterally over the fronto-parietal regions little or no air was seen. In the recumbent projections the asymmetry in the lateral ventricles persisted, again more obvious in the anterior portion. There was absence of subarachnoid air over the lateral aspect of the left hemisphere but with the midline sulci visualized and with the right side showing good demon-

stration of pathways. The temporal horns were clearly demonstrated on both sides and in normal position. Dilatation of the anterior portion of the left lateral ventricle indicated the existence of overlying cerebral atrophy. The lack of filling of the subarachnoid pathways over the outer aspect of this hemisphere suggested the probable existence of arachnoid adhesions. The neurological diagnosis was a thrombosis of the left middle cerebral artery probably resulting from poliomyelitis.

A few days after admission the patient's condition gradually began to improve. Therapeutical procedures consisted of radiant heat applied to the right extremities, upper and lower, for thirty minutes, followed by deep sedative massage. Rhythmic voluntary movements were carried out to improve coordination. The only speech therapy provided was that which was incidental to the testing procedures outlined in this report.

When first examined the patient seemed alert, well oriented and responsive to his environment. He was right-handed but exhibited a right-sided hemiparesis which prevented him from using his right hand for writing in the early stages of his convalescence. An audiometer test showed that his auditory acuity fell within the normal range but was slightly better for bone conduction than for air. He appeared to comprehend little that was said to him unless it was accompanied with appropriate gestures. He had only a few intelligible words for voluntary use but spoke a complicated jargon without seeming aware that it did not make sense. If one observed him speaking at a distance his intonation and syllabication seemed to have a normal rhythm; it was as if he spoke a foreign tongue. When spoken to he made a great effort to understand, scowled, squinted his eyes, and bent his ear toward the speaker; and by means of subtle gestures asked to have the phrase repeated. Occasionally he was able to repeat a word or two of what he heard and then seemed to comprehend better what had been said. After a few unsuccessful attempts he often expressed his frustration in mild profanity.

The subject was unable to carry out commands put to him in writing. Careful tests showed that he was able to recognize words well enough to match pairs but he was unable to interpret their meaning. All memory for the correct spelling of a word seemed lost when the patient attempted to write words to dictation with his left hand. Occasionally he wrote a semi-correct version of an unintended word. His spontaneous writing was jargon.

Although specific knowledge with regard to the location of the cortical lesion in this patient was lacking, the language findings were considered appropriate evidence for classifying him as a typical case of Wernicke's aphasia.

IV. Procedures

The six tests were arranged to be presented twice in a given testing period, first with the sounds or words being uttered by the examiner with her mouth hidden by a card, and second with the examiner's lips uncovered so that the subject could both see and hear the stimulus sound or word.

The subject sat at a table opposite the examiner who uttered the sounds or words in an average conversational tone. No exaggerated movements of the articulatory mechanism were employed to aid the subject in arriving at the correct response.

TEST I. Vowels, Semi-Vowels, and Diphthongs
(Time interval between stimulus sounds: 2 seconds)

Examiner's Lips Hidden			Examiner's Lips Uncovered		
Symbol	Response	Observation	Symbol	Response	Observation
[i]	+		[i]	+	
[ɪ]	+		[ɪ]	+	
[e]	+		[e]	+	
[ɛ]	[eɪ]	perseveration	[ɛ]	[eɪ]	perseveration
[æ]	+		[æ]	+	
[u]	+		[u]	+	
[o]	+		[o]	+	
[ɔ]	[oʊ]	perseveration	[ɔ]	[oʊ]	perseveration
[ɜ]	+		[ɜ]	+	
[ʌ]	+		[ʌ]	+	
[ɑ]	+		[ɑ]	+	
[aɪ]	+		[aɪ]	+	
[aʊ]	[oʊ]		[aʊ]	+	
[jɑ]	[ɑ]		[jɑ]	[eɪ]	
[ɔɪ]	[ɔ]		[ɔɪ]	+	
[ɛl]	[təl]		[ɛl]	+	

General Observations

1. Tendency to perseverate about equal with auditory stimulus alone and auditory plus visual stimulus.
2. Visual plus auditory stimulus more effective in reducing the number of incorrect responses with diphthongs than with pure vowels.

To keep the tests from becoming too complicated and endangering the results by fatigue or discouragement in the patient, it was decided to include only those symbols of the International Phonetic Alphabet which would ordinarily be required for the perception and emission of intelligible English speech. Wherever it was possible to do so, in the selection of stimulus words, concrete words of frequent occurrence in the speech of the average adult were employed.

The subject was permitted to observe the recording of his responses, which incidentally had a very stimulating effect

on his rapport in every testing situation. A correct response was recorded as plus; all other responses were transcribed into the International Phonetic Alphabet for later analysis.

TEST II. CONSONANTS INCLUDING AFFRICATES
(Time interval between stimulus sounds: 2 seconds)

Examiner's Lips Hidden			Examiner's Lips Uncovered		
Symbol	Response	Observation	Symbol	Response	Observation
[b]	+		[b]	+	
[m]	+		[m]	+	
[v]	[f]	confusion in voicing	[v]	[f]	confusion in voicing
[ð]	[mə]	see also [θ] below	[ð]	+	
[n]	[ʌ]		[n]	+	
[d]	[s]	see also [t] below	[d]	+	
[z]	[s]	confusion in voicing	[z]	[si]	confusion in voicing
[dʒ]	[ʃ]	confusion in voicing	[dʒ]	[ʃ]	confusion in voicing
[g]	[ʌ]	omission of gutteral	[g]	[ʌ]	see also [k] below
[p]	[ʌ]		[p]	[b]	confusion in voicing
[f]	+		[f]	+	
[θ]	[mə]	see also [ð] above	[θ]	+	
[s]	+		[s]	+	
[ʃ]	+		[ʃ]	+	
[t]	[sɪ]	see also [d] above	[t]	[d]	confusion in voicing
[tʃ]	[ʃ]	incomplete	[tʃ]	[ʃ]	incomplete
[k]	[h]	omission of gutteral	[k]	[ʌ]	see also [g] above

General Observations

1. Incorrect responses reduced about one-third when both auditory and visual stimulus provided.

2. There seems to be a tendency toward confusing surd and sonant consonants either in perception or initiation, or both.

3. The peculiar consistency observed in the subject's substitution of [mə] for [ð] and [θ], and also [s] for [d] and [t], is interesting.

4. More incorrect responses shown on consonant test as compared to vowel test.

5. The incorrect responses in this test appear to be about equally divided between the sonant and surd consonants.

Each symbol was uttered only once. At least a half-day interval was allowed between each test and the total testing period ranged over approximately three weeks.

Tests I and II consisted of isolated symbols of the International Phonetic Alphabet, vowels, semi-vowels, consonants and affricates. An interval of two seconds was allowed between the utterance of each stimulus sound. In these tests, as in the other four, all symbols were presented first with the auditory stimulus alone and then with both the auditory and visual cues.

Test III consisted of one-syllable words including vowels, sonant consonants and sonant affricates.

Test IV consisted of one-syllable words including vowels and at least one surd consonant or affricate.

Test V consisted of two-syllable words including only vowels and sonant consonants or affricates.

Test VI consisted of two-syllable words including vowels and one or two surd consonants or affricates.

TEST III-A. ONE-SYLLABLE WORDS: ALL VOICED SOUNDS
(Time interval between stimulus words: 2 seconds)

Examiner's Lips Hidden			Examiner's Lips Uncovered		
Symbol	Response	Observation	Symbol	Response	Observation
gun	[fɔr]		gun	[eɪl]	
ear	[i]		ear	[i]	
bell	+		bell	+	
mail	+		mail	+	
vine	[veɪl]	perseveration	vine	[feɪl]	perseveration
oar	[ɛl]	perseveration	oar	+	
the	[ɛl]	perseveration	the	+	
nose	[mɛl]	perseveration	nose	[ɛl]	
z	[si]	confusion in voicing	z	[si]	confusion in voicing
lad	[seɪl]	perseveration	lad	[ɛl]	
doll	[bɑl]		doll	+	
rouge	[si:ɛl]		rouge	[ʃi]	
you	[i:ɛl]	perseveration	you	+	

General Observations

1. Greater evidence of perseveration in the one-syllable words than in isolated sounds. Two-second time interval between stimulus words is probably not enough to eliminate the possibility of perseveration. Test should be repeated with time interval of at least 15 seconds. (See Test III-B.)
2. Great tendency to perseverate from words ending in [l].
3. Fewer incorrect responses when lip reading combined with auditory stimulus.

Much evidence of perseveration was manifested in the first administration of Test III, which suggested that the time interval between the stimulus words was too short, so instead of the established two-second interval between the stimulus words, an interval of 15 seconds was allowed. Test III was readministered on a succeeding day with favorable evidence toward the elimination of perseveration. A 15-second time interval between stimulus words was thenceforth employed in all later tests.

TEST III-B. ONE-SYLLABLE WORDS: ALL VOICED SOUNDS
(Time interval between stimulus words: 15 seconds)

Examiner's Lips Hidden			Examiner's Lips Uncovered		
Symbol	Response	Observation	Symbol	Response	Observation
gun	[ɛn]	omission of gutteral	gun	[ʌn]	omission of gutteral
ear	[hi]		ear	[eɪr]	
bell	+		bell	+	
mail	+		mail	+	
vine	[maɪn]	perseveration	vine	+	
the	[ðeɪ]		the	+	
nose	[ðouz]	perseveration	nose	[sou]	
z	[si]	confusion in voicing	z	[si]	confusion in voicing
lad	[seɪl]	perseveration	lad	+	
doll	[bɑl]		doll	[bɑl]	
rouge	[u]		rouge	[ʃi]	
you	[hu]		you	[hu]	

General Observations

1. Fewer perseverations are shown with the increase of time interval to 15 seconds between the stimulus words.
2. More completely correct responses are shown on the previous test, but the incorrect responses on this test show greater similarity to the stimulus word than in the previous test.
3. The gutteral sound [g] is omitted in the subject's responses in this test as in the previous one.

TEST IV. ONE-SYLLABLE WORDS INCLUDING SURD CONSONANTS
(Time interval between stimulus words: 15 seconds)

Examiner's Lips Hidden			Examiner's Lips Uncovered		
Symbol	Response	Observation	Symbol	Response	Observation
paint	[pɑɪ]	omission of final surd consonant	paint	[bɔɪ]	omission of final surd consonant
fence	[fɛn]	omission of final surd consonant	fence	[fɑl]	
tub	[bʌd]		tub	[sɑl]	perseveration
thumb	[hʌnt]		thumb	[θʌn]	
stove	[seɪl]	difficulty with affricate	stove	[sou]	difficulty with affricate
sheep	[seɪl]	perseveration	sheep	[ʃʌ]	
chick	[ʌ]		chick	[ʃɪkːn]	
cow	[hɑu]	omission of gutteral [k]	cow	[hɑu]	omission of gutteral [k]
horse	+		horse	+	

General Observations

1. Only two completely correct responses; incorrect responses show less similarity to stimulus words than in Test III.
2. Surd consonants in the final position usually omitted.
3. The patient expressed disgust at his inability to obtain more plus responses.

V. Summary and Conclusions

The present study involved an attempt to investigate the difficulties in perception in a case of Wernicke's aphasia. The approach to the problem was through a phonetic analysis of one patient's paraphasic speech. The speech samples were obtained through the patient's attempts to repeat after the examiner a series of isolated phonetic symbols and words. Under the conditions of the investigation the following observations were made; they may be significant for a better

TEST V. Two-Syllable Words Including Only Sonant Consonants
(Time interval between stimulus words: 15 seconds)

Examiner's Lips Hidden			Examiner's Lips Uncovered		
Symbol	Response	Observation	Symbol	Response	Observation
orange	[ɔr]	one-syllable response	orange	[ɔrn]	one-syllable response
robins	[ræzgo]		robins	[ræzgo]	
mother	+		mother	+	
iron	[aɪ]	one-syllable response	iron	[aɪə]	two-syllable response
wagon	+		wagon	+	"
baby	[mɛlmɪ]	two-syllable response	baby	+	"
zero	[sɪgə]	unvoicing [z]	zero	[sigə]	unvoicing
money	[meɪl]	one-syllable response	money	+	two-syllable response
very	[fɛr]	unvoicing	very	+	"
daddy	[dæf]	one-syllable response	daddy	[dɛf]	one-syllable response
even	[i:eɪ:ɛf]		even	+	two-syllable response
easy	[si]	one-syllable response	easy	[ɪr]	one-syllable response
waded	[weɪd]	one-syllable response	waded	+	two-syllable response

General Observations

1. No perseveration manifested.
2. More two-syllable responses with auditory and visual stimulus combined.
3. Two-syllable words with no surd consonants apparently cause little more difficulty than one-syllable words with surd consonants.

understanding of the difficulties in perception of the receptive dysphasic case that was studied, and possibly receptive dysphasics in general.

1. A considerable number of the subject's incorrect responses to the stimulus sounds or words bear enough resemblance to the test symbol to suggest that the subject was actually attempting to reproduce what he heard.

2. The greater number of correct reponses to simpler sound combinations, as in the case of the one-syllable words

including only sonant consonants, suggests, to the present investigator at least, that the subject's difficulties were predominantly those of perception rather than initiation.

3. Without the cue of lip reading, diphthongs appear to present greater difficulty than pure vowels.

4. Consonants appear to present more difficulty than vowels, and the advantages of lip reading seem to be more conspicuous with consonants than with vowels.

TEST VI. TWO-SYLLABLE WORDS INCLUDING SURD CONSONANTS
(Time interval between stimulus words: 15 seconds)

Examiner's Lips Hidden			Examiner's Lips Uncovered		
Symbol	Response	Observation	Symbol	Response	Observation
chickens	[ʃɪkɛn]	final consonant omitted	chickens	[ʃɪk]	one-syllable reponse
cookies	[ho]	one-syllable response	cookies	[hər]	one-syllable response
football	[bɔl]	one-syllable response	football	[halbam]	two-syllable response
spider	[mal]	perseveration	spider	[sɛl]	
rooster	[sou]		rooster	[su:ə]	
circus	[pɜs]		circus	[sɔr]	
fountain	[fant]	one-syllable response	fountain	[farn]	one-syllable response
pencil	[fɔrt]	perseveration	pencil	[mæl]	
scissors	[sɪdə]	two-syllable response	scissors	[sɛkɪmɛl]	
coffee	[soutɔr]	perseveration	coffee	[eɪ:ɛlmɛl]	
thistle	[fɔrt]		thistle	[θɪgɛl]	
polish	[fɔrt]	perseveration	polish	[mɛl]	
turkey	[hɜt]		turkey	[sɛlm]	

General Observations

1. No completely correct responses.
2. Incorrect responses seldom resemble stimulus word.
3. All but eight responses are in the form of one syllable.
4. Results suggest that two-syllable words including surd consonants present greater difficulties both in perception and initiation than do one- or two-syllable words including only sonant consonants.
5. Greater evidence of perseveration is shown than when the two-syllable words contain only sonant consonants.
6. Slight improvement toward a correct response with the additional cue of lip reading is evidenced in the subject's responses by the fact that the initial position of the stimulus word more closely approximates the correct one.

5. When consonants are presented in isolation the relative sonority or surdity of a given sound does not appear to influence the correctness of the response.

6. There appears to be a tendency toward confusion in voicing and unvoicing certain sounds.

7. Evidence of the tendency to perseverate on isolated *sounds* is very slight as compared to the greater incidence of perseveration on stimulus *words*.

8. A greater tendency toward perseveration was shown when the time interval between the stimulus words was two seconds than when it was 15 seconds, and also when two-syllable words including one or more surd consonants were presented.

9. There was generally less tendency toward perseveration when both the visual and the auditory stimulus were provided than when only the auditory stimulus was presented.

10. Both one- and two-syllable words including surd consonants obviously present greater difficulty, either in perception or initiation, than one- and two-syllable words including only sonant consonants.

11. There is a tendency for both surd and sonant consonants to be either incorrect or omitted when occurring in the final position in the stimulus word.

Implications of the observations in the present study suggest that acoustic training in verbal perception for receptive dysphasics may be more effective if it takes into consideration the level of difficulty represented by a given sound or combination of sounds. If the tendency toward perseveration is definitely related to the rate of utterance and also to the complexity of the sound combinations, these factors should be taken into consideration in organizing a plan of linguistic rehabilitation in receptive dysphasics.

REFERENCES

1. Hemphil, R. E., and Stengel, E. Pure word deafness. *J. Neurol. Psychiat.*, 1940, 3, 251-262.
2. Nielsen, J. M. Agnosia, apraxia, aphasia. Los Angeles: Los Angeles Neurol. Soc., 1936.

FORMAL CALL FOR PAPERS

American Speech Correction Association

1944 ANNUAL CONVENTION—DECEMBER 28, 29, 30

LASALLE HOTEL, CHICAGO

The Program Committee will receive for examination from now until the deadline, September 30, abstracts of papers and addresses proposed for a place on the program of the 1944 meeting of the Association. Three general types of papers will be considered:

1. Technical and scientific papers on speech pathology.
2. Papers dealing with inter-connections between speech correction and related fields.
3. Evaluations of the status of the speech correction profession, and its prospects for the future.

The Committee will operate under the rules set down in the Constitution of the Association.

All abstracts must be received by the Committee by September 30, 1944. In addition to summarizing the paper, each abstract must indicate (a) the time required for presentation, (b) details on the type of visual or audio aids which will be needed, and (c) other requirements. The full name and the professional affiliation of the author should be indicated.

Receipt of an abstract will be taken as evidence that the person submitting it is aware of the following rules governing the conduct of section meetings:

1. All meetings will begin when scheduled.
2. Chairmen will, without exception, hold speakers to time limits on papers, addresses and discussions.
3. As in the past, copies of all papers and addresses are to be collected by chairmen of section meetings, who will submit these manuscripts to the Editor of the *Journal of Speech Disorders*.

The Committee will welcome suggestions from members regarding non-members who can contribute significantly to the program. Such recommendations are to be transmitted to the Committee through a Fellow of the Association.

THE PROGRAM COMMITTEE

Margaret Letzter
Letitia Raubicheck
Harold Westlake
Raymond Carhart, *Chairman*
124 West Pearl Street
Butler, Pennsylvania

RHYTHMOKINESIS IN STUTTERERS AND NON-STUTTERERS

CHARLES R. STROTHER, Ph.D.
State University of Iowa
and
LOIS SMASON KRIEGMAN, M.A.
Institute for Juvenile Research, Chicago, Ill.

I. INTRODUCTION

During the past twenty years there has been a persistent search for neuro-physiological differences between stutterers and non-stutterers. A number of investigators have studied the rate of alternating movement (*diadochokinesis*) of various parts of the body musculature. The present authors (11) recently reviewed the principal studies of diadochokinesis and presented additional data which throw doubt on the existence of significant differences in diadochokinesis between stutterers and non-stutterers.

Another aspect of this problem, which has been studied by several investigators, is the possibility of differences in the rhythm of movement. Wertham (12) has demonstrated that disturbances in motor rhythm, which he terms *arrhythmokinesis*, may exist independently of adiadochokinesis in certain neurological diseases. The possibility of differences between stutterers and non-stutterers in rhythmic movement is, consequently, a subject of considerable theoretical interest.

In previous studies of this subject, rhythm has been defined in two different ways. Anderson (1), Blackburn (3) and Seth (9) have been concerned with variability in the rate of diadochokinetic movements. Anderson found no significant differences between stutterers and non-stutterers in variability in rate of foot-tapping. Blackburn obtained statistically significant differences in variability in the rate of movement of the diaphragm, tongue, lips and jaws, and no significant difference in variability of rate in hand-tapping. Seth repeated Blackburn's study with improved techniques. He obtained a significant difference in variability in rate of movement of the diaphragm and nonsignificant differences in variability of rate of movement of the lips and jaws.[1]

On the other hand, Wulff, (15), Cross (4) and Hunsley (5) have been interested in the ability to reproduce a given rhythmic pattern by movements of various parts of the musculature. Wulff investigated the rhythm of movement of the lips, jaws, tongue and hands and concluded that, on the average, stutterers are not inferior to non-stutterers in the ability to reproduce given temporal intervals. Cross, using the Motor Rhythm test of the Stanford Motor Skills battery, found no significant differences between stutterers and right-handed normals, and differences approaching significance (critical ratios of 2.6 and 2.5 for the left and right hands, respectively) between stutterers and left-handed normals. Hunsley investigated movements of the lips, jaws, tongue and thorax and found statistically significant differences between stutterers and non-stutterers with respect to the accuracy of reproduction of a given rhythmic pattern with movements of these structures.

[1]These differences obtained by Seth, however, were in the same direction as those obtained by Blackburn, with the stutterers inferior to non-stutterers.

II. Statement of the Problem

In the present study, we have been concerned with investigating the ability of stutterers and non-stutterers to reproduce a given rhythm pattern by movement of the lips, jaws, tongue and finger. The hypothesis to be tested is that stutterers do not differ significantly from non-stutterers with respect to the ability to reproduce a temporal rhythm by movement of these structures.

III. Subjects

The experimental group consisted of fifteen adult stutterers, eleven male and four female. A control group, obtained from the university population, was matched with the experimental group on the bases of age, sex, dextrality quotient on the Iowa Laterality Test, and rhythm discrimination on the Seashore test. The mean chronological age of the stutterers was 23 years; of the controls, 24 years. The mean dextrality quotient of the stutterers was 80; of the controls, 81. The mean rhythm score of the stutterers was 22; of the controls, 22.[2]

IV. Apparatus

For recording tongue movement, a face mask connected by a rubber tube to a Marey recording tambour was used. The subject repeated the *t* sound with the mask fitted tightly against the face.

For recording movement of the jaws, a rubber bulb fixed between two tongue depressors, which were held together by rubber bands at either end, was used.

In obtaining lip movement, a small rubber balloon was used. The tip of the balloon was placed between the subject's teeth and the apparatus was so arranged that unless the teeth were held together, the balloon would not remain between the lips. This arrangement effectively eliminated movement of the jaws, while obtaining a sensitive record of the lip movements.

For recording finger-tapping, a tambour with a small circular contact plate glued to the middle of the tambour diaphragm was used.

The stimulus pattern was produced by the same apparatus that had been used by Hunsley (5). It consisted of an aluminum disk fitted on a phonograph turntable. The edge of the disk was fitted with lugs, one inch in length. When the turntable revolved, the lugs came into contact with a stiff strip of brass, producing a series of audible clicks. The lugs were so spaced that the clicks came in a pattern of ¾ time, two eighth-beats followed by two quarter-beats. The subject reproduced the rhythm with the movements previously described. The speed of the turntable was set at 33 revolutions per minute and the speed was checked by a stroboscopic disk before each record, to insure a constant stimulus rate. The stimulus pattern was recorded simultaneously with the response pattern by a separate polygraph pen.

Records of the response patterns were obtained with a Marey recording tambour attached to a constant-speed polygraph.

V. Procedure

The subject was given the following directions: "You will now hear a pattern of four clicks. Listen carefully." The turntable was then started and five repetitions of the pattern were

[2]These groups were the same as those used in the study of diadochokinesis which has been reported previously (11). A detailed table showing the closeness of matching of each pair is included in the previous paper.

given, followed by these additional instructions: "I want you to reproduce this pattern as nearly perfectly as you can by tapping with your knuckles on the table, following both the pattern and the speed, like this—(two patterns demonstrated). Start on the second pattern—do not start on the first." After the subject had performed five patterns by tapping with the knuckles while the turntable was producing the stimulus pattern, he was asked to produce the *t* sound simultaneously with the tapping. Following this, he was given five more trials with the tongue alone, using the mask and following the pattern. Next, three trials of three patterns each were recorded. This general procedure was followed for each movement—five stimulus patterns produced while the subject merely listened to the rhythm, five more which he accompanied by tapping his knuckles, five patterns combining the knuckle-tapping and the movement to be recorded, five practice trials with the stimulus accompanied by the movement to be recorded, and finally three recorded trials of three patterns each.

Hunsley's procedure differed in several ways from that used in the present experiment. She introduced the stimulus pattern for a short time, then asked the subject to reproduce the pattern, after which a short practice period was given. Her directions then specified that the subject begin as soon as possible after a signal to begin was given. The pattern was to be repeated only once. The amount of practice was apparently not standardized. The method used in the present experiment provided a standard practice period for all subjects.

VI. Treatment of Data

Treatment of data of the type obtained here presents something of a problem. The record consists of a stimulus line, on which is shown the moment of occurrence of the four clicks that constitute the stimulus pattern; a response line, on which is shown the moment of occurrence of the movements that constitute the response; and a time line. At the polygraph speed which was used, the distance between the first two clicks of the stimulus pattern was 7 mm.; between the second and third clicks, 14 mm.; between the third and fourth clicks, 14 mm. Each movement in the response pattern might precede, coincide with, or follow its particular stimulus, and the total time occupied by the response pattern might be either greater or less than the time required for the total stimulus pattern.

Hunsley used a method of scoring which consisted in finding the ratios between the first (AB) and second (BC), and between the second (BC) and third (CD), time intervals of the performance pattern, determining the difference between each of these ratios and its corresponding ratio in the stimulus pattern, summing these values without regard to sign, and multiplying them by the ratio between the duration of the stimulus pattern ($A'D'$) and the duration of the response pattern (AD), in order to take into consideration differences in the duration of the total patterns. The formula was:

$$\left[\left(\left|\frac{AB}{BC} - \frac{A'B'}{B'C'}\right| + \left|\frac{BC}{CD} - \frac{B'C'}{C'D'}\right|\right)\frac{AD}{A'D'}\right]100$$

There are two criticisms of this procedure. In the first place, a performance which has the same ratios as the stimulus but which is longer or shorter than the stimulus pattern obtains a perfect score (a score of 0). A second, and much more important, objection lies in the unknown character of a distribution of ratio scores. There is at present no sufficient evidence to indicate that ratios such as these are distributed normally and, consequently, there is no justification for evaluating differences between such ratios by reference to normal probability theory.

A different procedure was adopted in treatment of the present data. Scores were obtained by determining the difference (in mm.) between each interval in the response pattern and the corresponding interval in the stimulus pattern, squaring each difference, and adding the squares. The formula[3] used was:

$$(AB-A'B')^2+(BC-B'C')^2+(CD-C'D')^2$$

The final score for each individual was the sum of these values for all three trials.

VII. Results

Table I shows the results obtained for the two groups.

TABLE I. Comparison of the Performance of Stutterers with that of Non-Stutterers in the Reproduction of a Rhythm Pattern by Movement of the Lips, Jaw, Tongue and Hand.

Movement	N	Mean	SD dist.	Diff.	d.f.	*t*	Level of Confidence
Lip							
Stut.	14	35.6*	18.7				
Non-S.	14	54.6	41.1				
				19.0	13	1.461	10-20%
Jaw							
Stut.	10**	16.1	4.7				
Non-S.	10	20.6	13.4				
				4.5	9	.849	40-50%
Tongue							
Stut.	14	26.1	15.3				
Non-S.	14	28.3	17.7				
				2.2	13	.452	60-70%
Finger							
Stut.	13	16.4	7.6				
Non-S.	13	18.2	8.9				
				1.8	12	.803	40-50%

* This figure represents the total score for three trials, derived from the formula given above. The lower the score, the more accurate the performance. A perfect performance would obtain a score of zero (0).

** The number of pairs differs for the different movements because of the exclusion of all records in which either member of a pair failed to respond with four movements in each of the three trials. Exclusion of such records was necessary because no valid way of scoring them could be devised. Since these errors were distributed approximately equally between stutterers and non-stutterers, their exclusion does not weight the results in either direction.

Inspection of Table I indicates that the stutterers were consistently, although not significantly, superior to non-stutterers on all movements. The magnitude of the SD's indicates that the individual differences among the non-stutterers were greater than the individual differences among the stutterers.

[3]This formula was suggested by Professor Lloyd Knowler of the Department of Mathematics, University of Iowa.

The difference between the variances on the lip and jaw movements was significant beyond the 1 per cent level, when examined by the *F* test. This makes the *t* values for these movements of questionable validity, since the *t* test involves the assumption of homogeneous variance in the populations from which the samples to be compared are drawn. However, since the large variances in the non-stutterers were produced by one extreme deviation in the case of the jaw movement, and two extreme deviations in the case of the lip movements, the use of samples of larger size would probably reduce both the variance and the mean of this group, thus reducing the magnitude of the difference between the two groups.

The data shown in Table I indicate that the stutterers are slightly, but not significantly, superior to non-stutterers with respect to the ability to reproduce a given rhythm pattern with the tongue and finger, and that they probably do not differ significantly from the non-stutterers with respect to reproduction of rhythm patterns with the lips and jaw. These data confirm those obtained by Wulff (15), but are not consistent with Hunsley's (5) results.[4]

VIII. Summary

Fifteen stutterers were matched with fifteen non-stuttering controls with respect to age, sex, laterality and rhythm discrimination. These two groups were compared as to the ability to reproduce a given rhythm pattern with movements of the jaws, lips, tongue and forefinger. While the stutterers were consistently slightly superior to the non-stutterers, none of the obtained differences was found to be statistically significant. No evidence of arrhythmokinesis among the stuttering group was obtained. The results differ from those obtained by Hunsley but corroborate those obtained by Wulff.

[4]In order to evaluate the possibility that the difference between the present results and those obtained by Hunsley might be due to difference in the methods of treating the data, Hunsley's formula was applied to the lip movement data obtained in the present experiment. By Hunsley's formula the mean for the stutterers was 35.06; for the non-stutterers, 37.04. The difference was 1.98; *t* was .302, which is significant only at the 70-80% level. Analysis indicates that Hunsley's formula tended to *minimize* inferior scores and consequently, if stutterers were inferior to non-stutterers, the difference would be more marked when the data are treated by the formula used in the present experiment than when treated by Hunsley's formula.

References

1. Anderson, L. O. Stuttering and allied disorders—an experimental investigation of underlying factors. *Comp. Psychol. Monogr.*, 1923, 1, 1-78.
2. Arps, George. Motor efficiency of elementary grade pupils in speech correction schools. *Hamburg Lehrtzg*, 1934, 13, 597-599.
3. Blackburn, William B. A study of voluntary movements of diaphragm, tongue, lips and jaw in stutterers and normal speakers. *Psychol. Monogr.*, 1931, 41, 1-13.

4. Cross, H. M. The motor capacities of stutterers. *Arch. Speech*, 1936, 1, 112-132.
5. Hunsley, Yuba. Dysintegration in the speech musculature of stutterers during the production of a non-vocal temporal pattern. *Psychol. Monogr.*, 1937, 49, 32-49.
6. Kiehn, E. Untersuchungen über die fahigheit zu feinabgemessenen Bewegungen (Feinmotorick) bei stammelnden, stotternden und normalen Volksschulern. *Vox*, Hamburg, 1935, 21, 32-35.
7. Lindquist, E. F. Statistical analysis in educational research. New York: Houghton Mifflin Company, 1940.
8. Rotter, J. B. A study of the motor integration of stutterers and non-stutterers. Unpublished Master's Thesis, Univ. Iowa, 1938.
9. Seth, G. An experimental study of the control of the mechanism of speech, and particularly respiration, in stuttering subjects. *Brit. J. Psychol.*, 1934, 24, 375-388.
10. Spriestersbach, D. Caryl. An exploratory study of the motility of the peripheral oral structures in relation to defective and superior consonant articulation. Unpublished Master's Thesis, Univ. Iowa, 1940.
11. Strother, Charles R., and Kriegman, Lois Smason. Diadochokinesis in stutterers and non-stutterers. *J. Speech Disorders*, 1943, 8, 323-335.
12. Wertham, F. I. A new sign of cerebellar disease. *J. nerv. ment. Dis.*, 1929, 69, 486-493.
13. West, Robert, and Nusbaum, Edward. A motor test for dysphemia. *Quart. J. Speech*, 1929, 15, 469-480.
14. Westphal, G. An experimental study of certain motor abilities of stutterers. *Child Developm.*, 1933, 4, 214-221.
15. Wulff, J. Lippen—Kiefer—Zungen und Handreaktionen auf Reizdarbietungen nach unterschiedlichen Zeitintervollen bei normalsprechenden und bei stotternden Kindern im alter von etwa 14 Jahren. *Vox*, Hamburg, 1935, 21, 40-45

STUTTERING: I. A CRITICAL REVIEW AND EVALUATION OF BIOCHEMICAL INVESTIGATIONS

HARRIS HILL, M.A.
Indiana University

I. INTRODUCTION

Many studies have considered the biochemistry and physiology of the stutterer. Judging from the interpretations made, these studies have, for the most part, been performed to support preferred contentions and not in the interest of discovering biochemical, physiological, or psychological principles. However, investigation in the field of stuttering generally has been productive of a very considerable body of knowledge, and it would seem that sufficient is known to permit certain interpretations.

If it were possible to collect the known crude data on stuttering, shorn of fanciful interpretations and based on the observed event of stuttering, if those data could be gathered and in some way organized, beginning perhaps, with fields of biochemistry, physiology, psychology, and psychopathology, with the addition of any other fields which would aid in objectively arranging findings, this writer believes that some general principles would be found.

The reason that such an approach would seem to be indicated, as any speech pathologist would agree, is that the data on stuttering are in disorganization and chaos. This confusion has been emphasized by Kopp (24), among others. He asserts that such a condition does not exist through lack of endeavor but because no theory or method has been sufficiently comprehensive to include all phases of stuttering. To gain the greatest good for the stutterer all knowledge of the phenomenon must be integrated. To do this a theory must stand on its own merits, not on tradition, and it must be sufficiently basic to embrace all the behaviors of stuttering, not merely isolated facets of the totality.

Recently several investigators (44, 38) have emphasized the importance of studying the total individual. They mean, of course, that stuttering as an entity does not exist, that when one is dealing with a specific set of factors which may influence behavior, such as biochemical, physiological, or psychological differentials, consideration of possibly related factors should not be excluded; that if the student is inclined to evaluate data from one of these fields as having causal significance this should not prevent his becoming aware of findings and interpretations in other fields. Yet the same people who demand that an organismic approach to stuttering be adopted immediately begin to dissect the individual, asserting that this or that structure or

organ controls behavior, that deep-seated neurophysiological biases exist which express themselves in varying degrees of disorganized behavior, or that the stutterer becomes fixated at some mythical anal or oral level of development, when actually all that can be maintained is that the particular organ or structure in question is essential to intact organismic activity, and that the disorganized behavior has been developed as a function of the complex whole.

A natural science of behavior (cf. 19), and any science that treats of behavior must deal with natural events if it is to maintain its status as a science, should treat of an event as it is observed to occur. When considering stuttering the event consists of a particular person, with a unique history, talking to someone about some object or condition in a specific situation. The application of instruments with resultant accumulation of crude data must be recognized as the level of first abstraction. However, when this occurs the event is no longer being dealt with in its actual situational context; the instrumental procedures have begun the process of dissection and isolation. Interest is now focused on what is occurring in the organism, with complete or nearly complete neglect of the situation, the stimulus objects of the situation, and the behavioral history of the individual.

Among the manipulative techniques applied to the investigation of stuttering those of biochemistry are, perhaps, the least subject to inaccuracy; within the present limits of chemical knowledge, body fluids can be analyzed and the results can be stated in quantitative form. Effects on the physiology of an organism resulting from a shift in the total amount of some substances or in the ratios of various substances have been ascertained, whereas in other cases certain effects were not ascribable to any known shift in total amount or ratio.

Studies which have compared body fluid constituents of stutterers with those of non-stutterers have often yielded conflicting results. This divergence, which must make conclusions tentative, would appear to depend upon the differentials of procedure in three areas: (1) the selection of subjects; (2) techniques of measurement; and (3) handling of the data obtained in (2).

Difficulties that have been encountered in the first area have ranged from the equating of control and experimental groups as to age, sex, body surface, medical history, and socioeconomic level, to adequate evaluation of the severity of speech disturbance.

Techniques of measurement have differed widely; diurnal and longer term changes have presented some problems. The behavior of the subject immediately prior to examination has presented and will continue to present influences which favor

error in making comparisons, yet two of the greatest sources of error are, of course, the procedures of obtaining samples, and the methods employed in analyses. For example, in connection with salivary pH, collection may be accomplished by expectoration, by sampling directly from the mouth, by means of suction discs, or readings may be taken directly by using indicator electrodes. Added to the inconsistencies resulting from differing sampling techniques those due to methods of analysis create further problems. Studies of pH illustrate this very well as do investigations concerning blood or plasma constituents where, in some instances, the total serum calcium has been estimated without regard to that proportion which was diffusable.

The third area, that designated as treatment of the data, affords opportunity for the appearance of personal bias; this may be intentional or non-intentional, but is usually, or preferably, attributed to unwitting behavior. It should be recognized, however, that artifacts may be created even though attention to this aspect during the last few years has contributed to rigor in statistical treatment.

It is not a primary objective of this paper to criticize the biochemical studies in the field of stuttering. Such a procedure would be rather profitless as the exact conditions of any study could not be ascertained. Rather, any criticisms will only be incidental to the presentation and integraton of reported material. Beginning with plasma and whole blood constituents, consideration will extend to lymph and cerebro-spinal fluid, urine, saliva, alveolar carbon dioxide content, and metabolism.

II. Plasma and Whole Blood Constituents

Johnson, *et al.* (18), reported in 1933 that the serum calcium of the stutterers they studied was in the high normal range, and although inorganic phosphorous was somewhat low in three samplings, findings on the remainder of the subjects were either normal or high normal, thus indicating that the serum calcium-inorganic phosphorous balance appeared normal. A similar condition was found in regard to serum calcium and serum potassium. On the other hand, high normal measurements are indicated by Kopp's work in 1934 (23). He reported that diffusable calcium and total calcium were higher in stutterers by 10.4 and 5.5 per cent, respectively, that inorganic phosphate was increased by 14.8 per cent and serum potassium lowered by 6.63 per cent. The calcium-phosphorous balance again might appear normal but for the discrepancy in the non-diffusable calcium fraction, which might indicate a slight rise in plasma protein or albumin instead of the decrease obtained in this investigation. The calcium-potassium ratio presented a shift, the former being higher and the latter lower than normal values. Karlin and Sobel (20) found a lower mean potassium

value, which was reported as insignificant, and some changes which were slight when considering other plasma constituents.

Serum globulin as considered in Kopp's study (23) is reported as being decreased by 22.7 per cent. Karlin and Sobel (20) found a slight decrease.

Kopp's value for total protein indicated a 12.6 per cent decrease, and Karlin and Sobel found a slight decrease, while Anderson and Whealdon (1) found no differences in the proteins which govern agglutination. Sugar was found to be increased 17.4 per cent by Kopp; Johnson, *et al.*, report a slight increase; Karlin and Sobel found a very slight increase in the mg–sugar ratio. According to Kopp, albumin was lowered 8.85 per cent, and according to Karlin and Sobel it was slightly increased. Apparently no one has found significant differences between stutterers and normal speakers in any of the other plasma constituents.

The above is not an exhaustive account of plasma studies; at least one report was not available for study, that of Kopp (22), 1933, while probably there are several more that have been published in other countries which are not at hand. An instance of the latter is Seeman's study of 1936 (35), the abstract of which reported that 88.4 per cent of 155 child stutterers and 84.6 of adult stutterers examined showed abnormal blood composition and humors. Another type of study which is of little profit in a review but which nevertheless must be included is that of Hogewind (17). He expresses the opinion that stutterers are in calcium deficit, yet gives no experimental basis for such a belief.

As the findings of biochemistry and their significance for organismic functioning are so complex and extensive, little can be accomplished concerning the role of plasma constituents, other than to point out a few possibilities. Rather than making a few extremely general statements concerning the various serum constituents it will be sufficient for the present to summarize briefly the role of calcium. One of the mineral metabolisms will be thus illustrated, thereby showing the significance that shifts in blood composition may have for behavior.

Ionic shifts that may affect neuromuscular irritability are usually considered to be changes in the following relationship:

$$\text{Irritability} = \frac{[Na^{+}] + [K^{+}]}{[Ca^{++}] + [Mg^{++}] + [H^{+}]}$$

If Na or K is increased, irritability is increased, while irritability is decreased by a rise in Ca, Mg or H; calcium decreases cell membrane permeability, sodium and potassium increase it (10, 175; 5, 119; 37, 157). That calcium is not always an inhibitor and that its equilibrium with other constituents is impor-

tant is well illustrated by the work of Bull (9). He induced anesthesia by injecting magnesium into vertebrates with calcium deficit. When a trace of Ca was injected the animals quickly awoke and returned to sensitivity. Another example of this effect is supplied when the heart is perfused with a high calcium content solution, for cardiac action will be stopped in systole, i. e., the heart will stop beating in a contracted state.

Relatively small changes in Ca or K ion concentration affect nerve function profoundly. There is considerable evidence that acetylcholine is an essential factor in transmission of the nerve impulse. No matter what structure is being innervated the action of acetylcholine is enhanced by potassium and lowered by calcium if shifts are not excessive (5, 119). The exact site of action of these chemicals is not well known but is thought to be the motor end-plate. The inhibitory effect of calcium in normal proportions and ratios is widespread. It appears to have a somewhat similar function to that of the sympathetic nervous system, while the action of potassium is similar to parasympathetic stimulation.

The solubility of Ca in plasma is dependent upon the protein concentration, CO_2 tension, plasma pH, total ionic strength, magnesium concentration and the inorganic phosphorous concentration, as stated by Cantarow (10, 181). McClean and Hastings (27) maintain that the albumin to globulin ratio, the magnesium and citrate concentration, and temperature all influence the ionization of calcium. In other words, blood calcium exists almost entirely in the plasma, is diffusable and non-diffusable. The former fraction being ionized is usually available for body functions and composes 45 to 60 per cent of the total serum calcium. The non-diffusable fraction is inert, and is chiefly in equilibrium with protein. It may be diminished when plasma protein concentration is lowered, while the concentration of diffusable calcium is the result of equilibrium between total calcium and the plasma proteins. Although the two forms may vary independently, they usually represent a rather stable equilibrium which appears to be controlled by the parathyroid glands. And recent work indicates that the parathyroid glands are controlled by a hormone of the anterior lobe of the pituitary (5, 1006).

In stuttering where a rather definite, general finding of heightened serum calcium is reported, neuromuscular irritability may be the product of other blood constituents, or if increased irritablity of tissues is induced by changes in Ca the change must necessarily be in that portion of the diffusable calcium held in equilibrium by some other plasma member.

This possiblity has been given consideration by McClean and Hastings (27). Although they do not give too much credence to the supposition, they state that while the greater part of diffusable calcium is ionized it may be partly bound to some citrate-like substance. Thus, even if the diffusable fraction of Ca were in the high normal range, this would not always indicate that all this calcium would be accessible to the tissues, and one basis for increased irritability might be established. Shohl (37, 136, ff.) asserts that "one must proceed with great caution in interpreting and evaluating the quantitative results of these [blood serum calcium] experiments," and, "Even in simple salt solutions there is considerable doubt as to the degree of activity of calcium. When other cations and anions are present the difficulties of interpretation at present are insurmountable."

The agents responsible for elevated serum calcium when pathological conditions are absent range from frank dietary variation, through chemical metabolic imbalances, to such behavioral influences as emotion and exercise. Altogether there has been much disagreement in regard to plasma calcium content in the psychoses and during affective states. Atkin (3) reported higher values in schizophrenia and in the manic phase of manic-depression, while he noted lower calcium values in the depressed phase. He also found higher values in epileptics. Farr and Hodes (2) report that one-third of the psychotics tested showed abnormal serum calcium, and this figure coincides with that obtained by Rothschild and Malamud (32) for cerebro-spinal fluid Ca on a group of manic-depressive cases. Molitch and Eccles (29) report no relation between serum calcium and juvenile delinquency. The findings of Laird (25) indicate that "wide changes in emotional activity are accompanied by greater than average variations in serum calcium." Low calcium is supposedly associated with moodiness and depression. Rich (33) expresses the opinion that the least excitable individuals tend to have the most acid urine and saliva, and the most excitable persons tend toward alkalinity or neutrality. Atkin shares the same view.

That there is usually marked depletion of plasma calcium in tetany appears to warrant little consideration here, except insofar as there may be a remote possibility that the stutterer is subject to mild, chronic latent tetany (36). A few of these possibilities were considered under neuromuscular irritability. It seems to be more relevant that muscular activity results in elevated plasmatic calcium. Keys and Adelson (21) showed conclusively that there is a marked rise in serum calcium during and following brief periods of severe work. A quotation from Penfield and Erickson (32, 349) in this regard is enlightening:

During and after the epileptic seizure there is apparently an increase of serum calcium (Fletcher and Peden) as well as a 34 per cent increase of serum phosphorous (Weil and Liebert). This change has been proved to be due to the tonic and clonic muscular contractions during the fit. Wolff and Goodell (1940) report a low calcium output in the interval between seizures indicating possibly a sluggish endogenous calcium metabolism in epileptic patients. However, this low output may indicate an attempt to restore calcium stores depleted during the seizure. Scott and Pigott (1936) found that hypocalcemia was uncommon in patients with epilepsy, while hypercalcemia (11-14 mg) was more usual.[1]

Cantarow (10) states that ingested or intravenously injected Ca increases the plasma value, which returns to normal in from one to four hours. Thus if determinations were made on stutterers in the basal condition, elevated plasma Ca due to maintenance of tone or muscular contractions would probably have disappeared if chronic tensions did not exist.

The question of stutterers having heightened irritability of neuromuscular tissue cannot, of course, be settled here. But the answer to the question of increased plasma calcium appears to be very clearly indicated. The interpretation seems inescapable that if the stutterer's calcium intake is adequate and not excessive, and if no pathologies are present, the increase in plasma calcium is a direct reflection of activity in skeletal and smooth muscles. The modification of this statement that might be forced would be that brought forward by Kopp (23) when he maintained that the affective condition of the stutterer is not adequately indicated by the biochemical measures (i. e., compensation may obtain if affective states become chronic).

The red blood count and volume, and hemoglobin content of hematocritic determinations on stutterers have been shown by Trumper (45) to range from rather low to high normal, depending in nearly all cases upon the efficiency of respiratory functioning and the per cent absorption of inhaled oxygen. All his determinations were made under conditions appropriate for basal metabolism. Consequently these measurements would indicate only trends which were rather constant if there were no pathologies present or no transitory factors operating such as temporarily increased thyroid activity.

Twitmyer (47) in 1930 quoted Trumper extensively in regard to his findings on increased hemoglobin and R. B. C. count, and made the statement that these increases in shallow breathing stutterers are similar to those resulting from known pathological conditions.

The report of Kopp (23) indicates that his investigations point to a similar increase as shown above but he mentions other causal factors, such as affective states increasing the R. B. C. count, etc. He believes that due to compensatory

[1]References will be found in Penfield and Erickson (31).

mechanisms the degree of emotionality is not accurately reflected in the adult stutterer by blood sugar content, salivary pH. or R. B. C. count.

Blood count of stutterers was found to fall within the normal range by Card (11). The white cell count apparently was high normal. Trumper (45) had previously reported similar findings on some of his stuttering subjects.

Erythrocyte and hemoglobin changes are intimately correlated with oxygen tension and demand. Bard (5, 307) indicates that R. B. C. production is increased if oxygen supply is decreased, no matter how this decrease is induced. Although the increased demand for oxygen during exercise is chiefly met by decreased circulation time (i. e., cardiac output per minute is increased), there is an increase in R. B. C. count and volume (5, 309; 12, 100; 45, 29). Trumper (45) reported increases during fright, Kopp (23) implies that affective states may be somewhat reflected in the R. B. C. count, and Crafts (12, 100) shows that so-called emotional states produce decided increases, but that in seven minutes the count may return to normal.

Exercise and affect increase the leucocyte count (12, 101), and there appears to be a relation between amount of activity and this increase (5, 310). That factors other than increased muscular activity, such as hormonal influences, may be operative in affect is possible. Infections of any nature elevate the number of white blood cells, and the ingestion of various foods may cause a temporary rise.

That the W. B. C. count of stutterers may be somewhat above normal has been indicated by Trumper (45), Card (11), and Kopp (23). The agent responsible for the abnormality, if one exists, is maintained by the present writer to be the same as that involved in the R. B. C. and hemoglobin changes, and calcium increase (i. e., the normal physiological processes which occur during affect and muscular activity).

To arrive at a satisfactory conclusion, determinations should be made on stutterers during the basal state, in situations where little or no stuttering and affect are present, and during and after severe stuttering. In conjunction with these experiments, studies should be made of so-called normal speakers during periods of muscular work and under affective conditions.

III. Oxygen and Carbon Dioxide

Considerable difference of opinion has arisen in regard to CO_2 content and tension of alveolar air, and venous and arterial blood of stutterers. Some investigators postulate that all stutterers are either suffering from, or bordering on, anoxemia. Apparently these interpretations are drawn from carbon dioxide determinations and not directly from oxygen measure-

ments. Usually these two vary inversely; hemoglobin loses its affinity for O_2 as the CO_2 tension increases. This does not necessarily mean that a high CO_2 tension indicates anoxemia for other factors may operate to compensate for the increase, e. g., erythrocyte count and hemoglobin volume per cent may increase.

Starr (41) indicated that 80 per cent of his stutterers were sub-breathers, and were lethargic due to excessive CO_2; determinations were made on alveolar air. He noted that normal speakers tend toward low CO_2 tension, stutterers toward the high upper normal limits, and cured stutterers ranged between these groups. These findings may have been partially due to the time of day in which the measurements were made.

Trumper (45) did not deal directly with tensions or volumes of O_2 and CO_2. He reported that stutterers were generally in a state of mild anoxemia due to shallow breathing and inefficient O_2 exchange. In all cases where he found such conditions, what he termed hemato-respiratory compensation existed; inefficient respiratory reactions were partially compensated for "by increasing the red cells, red cell volume and the hemoglobin content of the blood." The normal standards of comparison that he used were what are usually called "textbook" norms, and all samplings were taken during basal conditions. The former might partly invalidate his findings and the latter would mainly indicate only conditions which were chronic.

Starr (41) reported sub-breathing and hyperexcitable stammerers as having high and low CO_2 tensions respectively, while Kopp (22) found no differences in alveolar air measurements between stutterers and normal speakers.

Basal metabolism of stutterers has been investigated by but a few people. From Trumper's (45) graphs and tables it is gathered that those adult subjects showing high R. B. C. count and volume tended toward higher B. M. R.'s than those stutterers with lower values. Ritzman (34) found non-significant differences between his groups of men, stutterers and normals, while "Differences with *t*'s ranking at the 5 per cent level were obtained between the women stutterers and their controls, and between the 'severe' stutterers and their controls."

In man, due to chemical and physiological equilibrium, excess CO_2 is compensated for by various mechanisms; some of these have been mentioned, some have not. So it would seem appropriate, as Trumper (45) postulated, that if the stutterer goes into oxygen debt, or accumulates an excess of CO_2, hemato-respiratory compensation would occur. That the stutterer has a chronically high alveolar air CO_2 tension, and consequently a like condition of arterial blood, has not

been proved. In fact, rather definite proof that stutterers as a class do not differ from normals in this respect has been offered by Kopp (22).

One possibility is that the stutterer during difficult speech, and the severe stutterer who is chronically affective, may have respiratory inefficiency; sub-breathing even at sea level results in anoxia (5, 687), and this may also occur in neurasthenia. Oxygen lack does not affect respiration until it reaches the 18 per cent level, so an effective anoxic stimulus for respiration could not be expected if the oxygenation of the blood is at all within normal limits. Muscle and nerve fatigue occurs more readily in anoxia, and the behavior of a person suffering from chronic anoxia resembles that of the neurotic, viz., constant fatigue, affective variability, muscular twitching, loss of self-confidence, and perseverative tendencies, according to Bard (5, 684). This text, however, states that no evidence has been found to show that lack of oxygen is responsible for these aberrant behaviors in neurotics.

Some severe stutterers possibly have heightened arterial CO_2 tension which may contribute to sluggish circulation and general lethargy. This is indicated by Ritzman's (34) findings of a lower basal metabolism rate in his severe stutterers than in their normal speaking controls. However, as indicated by Ritzman, conclusions should be tentative as the severe stutterers presented a mean B. M. R. of —16.43 as against —12.46 for all male controls. Yet that stutterers as a class are in a condition of CO_2 excess is very questionable.

IV. Urinary Creatinine

Stratton (42), in studying a stutterer over an extended period, reported that a rise in the creatinine excretion level was shortly accompanied by improved speech, while a lowering of urinary creatinine was followed by poor speech. Periods of excitement were followed by difficult speech, thus implying that during these periods the value of excreted creatinine fell.

Studies involving one subject are important, and perhaps more of them should be conducted, yet it is impossible to make any generalizations on reports of one subject. The most that can be done is to mention briefly several of the studies dealing with creatinine.

The output of this product of nitrogenous metabolism is said by Bard (5, 803) to be constant for any one individual, although the quantities excreted vary from person to person, being mainly dependent upon weight. The creatinine coefficient is lower for women than for men. This difference depends "on the relative degree of muscular development in the two sexes" (5, 804).

Rich (33), after studying creatinine in the human body, came to the conclusion that affectivity bears an inverse rela-

tionship to creatinine in the blood and urine, while Bowman (7) noted that creatinine and creatine are essentially normal in the psychoses, and Mittelshtedt and Novakovskaya (28) found creatine in the urine after producing emotional disturbances in their subjects.

V. Lymph, Cerebro-spinal, and Interstitial Fluids

As far as this writer knows there have been no attempts to analyze lymph, cerebro-spinal, or interstitial fluid of stutterers. Berry (6) reports that in examining two groups of children, about 500 normal speakers and 500 stutterers, she found that diseases of the lymphatic systems occurred in 4.42 per cent of the stuttering cases, and in .43 per cent of the non-stuttering cases. Normally calcium is in somewhat lower concentration in lymph than in plasma; investigation of lymph and interstitial fluid might throw some light on the problem of whether the tissues of stutterers are in a condition of accessible calcium deficit. Cerebro-spinal fluid calcium content in normals is placed at approximately half the plasma value; nevertheless there is a strong barrier to calcium entering the C. S. F. when shifts occur in plasma content. Here, again, investigation on stutterers might be of interest.

VI. Salivary pH

Recent work on salivary pH values of stutterers was done by Hafford in 1941 (15). She presented a strong disagreement with the findings of Starr (39, 40), who reported in 1922 that sub-breathing stammerers as a class had low salivary pH values and high alveolar air CO_2 content, while hyperexcitable psychopathic stammerers had high salivary pH and low alveolar CO_2 content in comparison with a normal control group. (Just how the groups could be so neatly parcelled off is not too clear.) This same investigator in 1928 (41) found blood pH to be lower and CO_2 content higher in sub-breathing stammerers than in their controls. The interpretation made by Starr was that salivary pH, together with salivary CO_2 value, might be taken as an index of fatigue and breathing habits, the degree of emotional excitement, and the excitability of the subject.

As far as Kopp (23) could determine in 1934, there were no significant differences between blood pH and CO_2 content of stutterers and his normal speaking controls.

Baker and Eye (4) checked and corroborated Starr's work in regard to salivary pH decrease during deep breathing, and during exercise, followed by a decrease upon cessation of exercise. Among their independent findings were: characteristic high pH during rest, slight drop during noise stimulation, immediate decrease in pH following a problem period. They were of the opinion that CO_2 seems to be the prime factor in control of salivary pH, but that there is lack of cor-

respondence between salivary and blood pH and pulmonary ventilation.

In 1938, Winsor and Korchin (48), following a study of parotid secretion, reported that there is a positive relationship between salivary pH value and the rate of flow. They did not find a consistent, direct relationship between parotid pH and pulmonary ventilation, or alveolar CO_2. However, they did verify Starr's results on fatigue; pH was more acid during this condition than when the individual was not fatigued. Also, emotional states were shown to be accompanied by inhibited salivary secretion, and this latter with low pH and dental caries.

Hafford's work (15), as mentioned, is the most recent relevant study on salivary pH that was inspected. Her determinations were on mixed saliva, taken under the tongue. This was done partly because saliva in this position may be that portion which is chiefly affected by speech, and partly because the sublingual gland secretes less intermittently than either the parotid or submaxillary. Under the rigorous conditions of her experiment, the findings show clearly that before speech activation there were no significant differences between the mixed salivary pH of stutterers and their normal speaking controls. Some differences between pH values of stutterers and non-stutterers were obtained directly following speech in which the stutterers had presented some blocking behavior; these differences were not statistically significant but indicated trends.

The conditions of this experiment were such as to insure optimal value secretion, i. e., aside from those determinations taken after speech activation, measurements under these conditions would show only those differences in pH secretion which were rather stable.

Except insofar as it is necessary for purposes of later integration, factors influencing secretion and pH of saliva will not be mentioned, as they have been rather well covered by the above authors. Differences have been found between stutterers' and normal speakers' salivary pH, but differences have also been found between normal speakers in this regard, and, also, in the same individual under different circumstances. It would seem evident that unless some pathology exists, changes in pH are due to the behavioral situation of the individual. In this connection it might be well to consider the statement advanced by Bard (5) that the rate of flow is almost entirely dependent upon parasympathetic excitation through vasodilatation channels, while pH values are lowered and the flow nearly stopped if sympathetic stimulation is dominant.

This picture is comparatively simple, but it might hold if the reaction were purely biological. That behaviorally ac-

quired reactions of secretion must be considered was early demonstrated by Pavlov (30), and in 1942 Finesinger, *et al.* (13) obtained the positive conditional salivary reflex in all psychoneurotic patients tested. Hafford (15) rather definitely showed that there are no differences between the resting saliva of stutterers and normal speakers, but she did not show that extreme changes in pH do not accompany prolonged periods of stuttering. Very probably this is where the greater part of her differences with Starr's (40) findings lie, for his determinations were apparently not made in the basal state. Starr's groups appear to be slightly too convenient; however, his report that 80 per cent of the stutterers show low pH values and that fatigue and exercise are accompanied by lowered values probably are significant. Winsor and Korchin (48) maintain that fatigue and emotional states are accompanied by low pH, Baker and Eye (4) say a similar condition exists following a period of mental work, while Winsor and Korchin (49) in their 1940 study, report that auditory stimulation, eye strain during reading, and protopathic stimulation of the skin reduce salivary secretion.

If the negative findings could be excluded it would seem that a clear-cut case had been established for the stutterer being an affective person whose salivary pH values changed with muscular effort and affectivity. However, Penfield and Erickson (31, 75) indicate that salivary flow is increased during epileptic seizures, Strongin and Hinsie (43) that pH values drop during the depressive phase of manic-depression, and Laurie, Barrera, and Strongin (26) note that of the behavior problem children studied whose secretion rate remained very high, none evidenced any improvement.

Just what occurs in the intact human organism functioning in a situation to which it has previously responded affectively is indeed problematic. Conditioning of behavior may influence the functioning of glands, as being a part of the total organismic response, to the point where autonomic stimulation is only one of the controlling influences. In regard to behaviors which are indicative of affect Gaskill and Cox (14) complete their paper with: "Our conclusion definitely is that emotional patterns are most highly variable, and that they are made up of many variates: A plain indicator of emotion does not exist."

VII. Summary and Conclusions

There may be some slight biochemical differences between the stutterer and the non-stutterer. Irritability of tissues may be increased in the stutterer even though blood calcium and phosphorous values appear to be somewhat elevated. Although a trend is seen toward high red and white blood cell count these tendencies are not constant. Investigation of alveolar air CO_2 has brought conflicting results: In some

instances carbon dioxide tension was found to be increased, whereas in other well controlled studies, in which determinations were made in the basal state, no differences from normal were reported. The facts of the case were stated as probably lying in favor of both reports, i. e., except for severe stutterers in which CO_2 tension differences may be found in the basal state, alveolar CO_2 tension probably is normal in the basal condition and somewhat abnormal following prolonged periods of stuttering. Basal metabolic rates, except those for severe stutterers and female stutterers, appear normal, and the close approximation of the female to the male stutterer's B. M. R. is thought to be due to increased activity. Elevations in the excretion of creatinine in the urine are reported. Following examination of one stutterer, fluctuations in the urinary excretion of creatinine are reported as being parallel to changes in speech performance, an increase being concomitant with less stuttering. Salivary pH values for the average stutterer were shown to be normal in the basal state with, possibly, a trend downward during the day's activity.

In all instances where differences between stutterers and normal speakers purportedly obtained, the differences were compared with and shown to approximate closely changes in so-called normal persons during muscular activity and affective behavior.

Interpretations given to findings on body fluids in stutterers have ranged from the assertion that they are determiners of all activity, whether physiological or psychological, to the more conservative view that body fluids, while participating in the maintenance of the organism and influencing its functioning, can only place limits on behavior and cannot be determiners of it. Naturally if body fluids were abnormal they would influence behavior, even to the point of death, but they could not create behavior. Such conditions might increase neuromuscular irritability and thus elicit physiological spasms and convulsions, yet activity of this nature which is induced by bodily changes is relatively invariable and will always result if certain conditions obtain. Molitch and Eccles (29) conclude their paper by saying that there is no justification in assuming that one chemical body has a relation to irritability of peripheral structures and a similar relation to the whole nervous system and to gross behavior of an individual. To be determiners of behavior, shifts in body fluids would necessarily have to be responsible for actions which cannot be placed in a structure-function framework (i. e., psychological behavior).[2]

[2]Consider the ontogenetic development of language as illustrative. Any language is learned through acquiring shared responses of a particular culture. The biochemistry of a particular individual may limit or influence the acquirement but does not determine the language that will be learned. All psychological phenomena can be dealt with in a similar manner.

Hypotheses concerning an affective or emotional genesis of stuttering are not new. This writer is well aware of the rather hoary discussion as to whether the affective behavior of the stutterer follows or stands in causal relation to stuttering. However, it would seem apparent that unless recourse is taken to non-naturals all organismic changes which the stutterer presents can only be interpreted as being the result of behavior which he performs. If we do not wish to resort to internal principles as determiners of psychological behavior—and we certainly do not—the conclusion is practically forced that biochemical differences between stutterers and non-stutterers are coincident with differences in affective and muscular activity. And, although the resultant changes may influence behavior through increasing or decreasing irritability, thus acting as stimuli in S⟶R functioning, they cannot be construed as the direct causal antecedents of psychlogical activity. Biochemical changes envisaged, in the main, as results instead of causes might be acceptable to most persons familiar with stuttering and the literature thereof, yet the proposition that the behaviors termed stuttering are developed through affective and emotional channels would, very probably, be unacceptable. Established attitudes, opinions, and beliefs would possibly be the primary obstacle to the acceptance of such a view, while the seeming impossibility of doing justice to all the phenomena of stuttering on these grounds might be only secondary.

Many questions immediately arise. For instance, such questions as to what is the stuttering block, what is its origin, and how does it attain such automaticity as it appears to exhibit, are but a very few that are pertinent. Obviously the answers do not lie in merely classifying stuttering as affective behavior; in any case these cannot be considered in this critical review of biochemical studies. An attempt at answering several questions has been made by this writer **(16)** and further investigations are in progress.

In concluding this discussion of findings on stuttering behavior it is well to point out definitely that this writer is in agreement with Brown (8) when he maintains that stuttering occurs on the basis of the normal functioning of a normal neurophysiological mechanism. Insofar as Brown builds his constructions upon Cannon's conceptions of emotions they would not be acceptable, because the emphasis is not upon organismic activity in specific situations; yet his emphasis is well placed when he asserts that it would appear entirely unnecessary to invoke abnormal structures, or functioning, as causal. In the case of biochemical differences no findings warrant any assumption of special metabolic or chemical agents which are causal. The phenomena of stuttering can well be

explained if principles of normal behavior are adhered to without attempting to make the stutterer a unique animal in the universe.

REFERENCES

1. Anderson, J., and Whealdon, M. L. A study of blood group distribution among stutterers. *J. Speech Disorders*, 1940, **6**, 23-28.
2. Appel, K. E., Farr, C. B., and Hodes, P. J. Chap. 9 *in* Manic-depressive psychoses, Vol. 11, *Res. Publ. Ass. nerv. ment. Dis.*, 1930. Baltimore: Williams & Wilkins, 1931.
3. Atkin, I. Serum calcium in the psychoses. *Lancet*, 1937, **232**, (I), 439-440.
4. Baker, K. H., and Eye, M. G. The reliability of the pH of human mixed saliva as an indicator of physiological changes accompanying behavior. *Amer. J. Psychol.*, 1935, **47**, 222-240.
5. Bard, P. MacLeod's physiology in modern medicine. St. Louis: Mosby Co., 1941.
6. Berry, M. F. Medical history of stuttering children. *Speech Monogr.*, 1938, **5**, 95-114.
7. Bowman, K. M. Blood chemistry in mental diseases. *Amer. J. Psychiat.*, 1923, **2**, 379.
8. Brown, F. W. Stuttering: Its neurophysiological basis and probable causation. *Amer. J. Orthopsychiat.*, 1932, **2**, 628.
9. Bull, H. B. Physical chemistry. New York: Wiley & Son, 1943.
10. Cantarow, A. Chap. 5, "Mineral metabolism," *in* Diseases of metabolism (G. G. Duncan, ed.). Philadelphia: Saunders Co., 1942.
11. Card, R. E. A study of allergy in relation to stuttering. *J. Speech Disorders*, 1939, **4**, 223-230.
12. Crafts, L. W., Schneirla, T. C., Robinson, E. E., and Gilbert, R. W. Recent experiments in psychology. New York: McGraw-Hill, 1938.
13. Finesinger, J. E., Sutherland, G. F., and McGuire, F. F. The positive conditional salivary reflex in psychoneurotic patients. *Amer. J. Psychiat.*, 1942, **99**, 61-74.
14. Gaskill, H. V., and Cox, G. M. Patterns in emotional reactions: II. Heart rate and blood pressure. *J. gen. Psychol.*, 1941, **24**, 409-421.
15. Hafford, J. A. A comparative study of the salivary pH of the normal speaker and stutterer. *J. Speech Disorders*, 1941, **6**, 173-184.
16. Hill, H. An interbehavioral analysis of several aspects of stuttering. *J. gen. Psychol.* In press.
17. Hogewind, F. Medical treatment of stuttering. *J. Speech Disorders*, 1940, **5**, 203-208.
18. Johnson, W., Stearns, G., and Warweg, E. Chemical factors and the stuttering spasm. *Quart. J. Speech*, 1933, **19**, 409.
19. Kantor, J. R. Survey of the science of psychology. Bloomington, Ind.: Principia Press, 1933.
20. Karlin, J. W., and Sobel, A. E. A comparative study of the blood chemistry of stutterers and non-stutterers. *Speech Monogr.*, 1940, **7**, 75-84.
21. Keys, A., and Adelson, L. Calcium changes in the plasma resulting from brief severe work and the question as to the permeability of the capillaries to calcium. *Amer. J. Physiol.*, 1936, **115**, 539-547.
22. Kopp, G. A. The metabolism of the stutterer as evidenced by biochemical studies of blood, alveolar air, and urine. Unpublished doctoral dissertation, Univ. Wis., 1933.
23. ————. Metabolic studies of stutterers: I. Biochemical study of blood composition. *Speech Monogr.*, 1934, **1**, 117-132.
24. ————. Treatment of stutterers. *Proc. Amer. Speech Corr. Ass.*, 1939, 1-8.
25. Laird. D. A., and Steffan, J. M. The relation of emotional tone to blood calcium. *Med. J. and Rec.*, 1933, **138**, 223-224.

26. Laurie, R. S., Barrera, S. E., and Strongin, E. I. Autonomic nervous system function in children with behavior problems as measured by the parotid secretory rate. *Amer. J. Psychiat.*, 1942, **99**, 419-425.
27. McClean, F. C., and Hastings, A. B. The state of calcium in the fluids of the body. *J. biol. Chem.*, 1935, **108**, 285-322.
28. Mittelshtedt, A. A., and Novakovskaya, E. S. Changes in metabolism in certain cases of emotional stimulation. *Arkh. biol. Nauk*, 1937, **45**, 119-126.
29. Molitch, M., and Eccles, A. K. Serum calcium in juvenile delinquents. *Amer. J. Orthopsychiat.*, 1934, **4**, 73-78.
30. Pavlov, I. P. Conditioned reflexes. An investigation of the physiological activity of the cerebral cortex. London: Oxford Univ. Press, 1927.
31. Penfield, W., and Erickson, T. C. Epilepsy and cerebral localization. Baltimore: C. Thomas, 1941.
32. Rothschild, D., and Malamud, W. Chap. 8 *in* Manic-depressive psychoses, Vol. 11, *Res. Publ. Ass. nerv. ment. Dis.*, 1930. Baltimore: Williams & Wilkins, 1931.
33. Rich, G. L. A biochemical approach to the study of personality. *J. abnorm. soc. Psychol.*, 1928, **23**, 158-175.
34. Ritzman, C. H. A comparative cariovascular and metabolic study of stutterers and non-stutterers. *J. Speech Disorders*, 1942, **7**, 367-373.
35. Seeman, M. Contribution to the pathogenesis of stuttering. *Rev. Neurol. Psychiat.*, Praha, 1936, **33**, 399-404.
36. Shackson, R. An action current study of muscle contraction latency with special reference to latent tetany in stutterers. *Arch. Speech*, 1936, **1**, 87-111.
37. Shohl, A. T. Mineral metabolism. New York: Reinhold Corp., 1939.
38. Simon, C. Integrated behavior in the stutterer and non-stutterers. *Proc. Amer. Speech Corr. Ass.*, 1936, **6**, 121-126.
39. Starr, H. E. Hydrogen-ion concentration in saliva, etc., as an index of fatigue and of emotional excitation, and applied to a study of the metabolic etiology of stammering. *Amer. J. Psychol.*, 1922, **33**, 394.
40. ————. Biochemical studies of human mixed saliva. *J. biol. Chem.*, 1922, **54**, 43-64.
41. ————. Psychological concomitants of high alveolar carbon dioxide; a psychobiochemical study of the etiology of stammering. *Psychol. Clin.*, 1928, **17**, 1-12.
42. Stratton, L. D. A factor in the etiology of a sub-breathing stammerer; metabolism as indicated by urinary creatine and creatinine. *J. comp. Psychol.*, 1924, **4**, 325.
43. Strongin, E. I., and Hinsie, L. E. A laboratory method for diagnosing manic-depressive depression. *Psychol. Bull.*, 1942, **39**, 509.
44. Travis, L. E. Laboratory research of the speech pathologist. *Proc. Amer. Speech Corr. Ass.*, 1938, **8**, 28-30.
45. Trumper, M. A. A hemato-respiratory study of 101 consecutive cases of stammering. Unpublished doctoral dissertation, Univ. Penn., 1928.
46. ————. The respiratory significance of blood counts. *Psychol. Clin.*, 1930, **19**, 7-12.
47. Twitmyer, E. B. Stammering in relation to hemo-respiratory factors. *Quart. J. Speech*, 1930, **26**, 278-283.
48. Winsor, A. L., and Korchin, B. The effect of different types of stimulation upon the pH of human parotid secretion. *J. exp. Psychol.*, 1938, **23**, 62-79.
49. Winsor, A. L., and Korchin, B. Some observations on the effect of mental activity upon parotid secretion. *J. gen. Psychol.*, 1940, **22**, 25-32.

A S C A
News and Announcements

MIRIAM D. PAULS, *Editor*
Indiana State Teachers College

The twenty-ninth annual meeting of the Acoustical Society of America was held in New York City, May 12 and 13, 1944. The first day was devoted to a symposium on the correction and prevention of hearing impairment. The chairman of the symposium was Dr. Harvey Fletcher. Fifteen papers were read on various phases of the topic. Capt. E. H. Truex, U. S. Army, Deshon Hospital, read a paper which outlined the Army aural rehabilitation program. Dr. Hallowell Davis reported on articulation tests for hearing aids. This is one of the projects being done at the Psycho-Acoustic Laboratory at Harvard. Capt. C. W. Schilling (MC), U. S. Navy, discussed acoustical trauma as encountered in submarine duty. Dr. Norman Watson presented a paper on an air and bone conduction audio-testing assembly. This apparatus is designed to obviate the use of a sound-proof booth in giving air and bone conduction audiometric tests.

Dr. Paul Sabine reported measurements of the over-all acoustic gain shown by six commercial hearing aids. This study shows that only one of the instruments tested showed any significant change in frequency response with change in the "tone control setting" of the instrument. Miss Miriam D. Pauls read a paper, "The Role of the College and University Hearing Clinic." (It is included in this issue of the *Journal.*) Dr. John C. Steinberg presented a paper entitled, "The Physics of Hearing Measurements." Mr. Howard Carter discussed the consideration of hearing aids and audiometers by the Council on Physical Therapy, an amusing portrayal of the problems encountered in advising on the accuracy of advertising.

The program on Saturday was a symposium on the use of sound waves in the study of liquids and solids.

At the University of Redlands, Dr. Conrad Wedburg has developed a course in military speech for officer training, designed to train officer candidates to lead and command more efficiently. An accelerated class, it met four times weekly: twice on the drill field and twice in the classroom. The course was considered a laboratory for self-improvement and workshop procedures were employed. Each student was trained to use his vocal mechanism effectively without strain; to give distinguishable, clear, accurate commands from a distance of one hundred yards; to read reports that could be understood. Officers given such training will be better prepared to avoid regrettable accidents caused by carelessly articulated commands.

(Continued on Page 272)

A COMPARATIVE STUDY OF LOUDNESS, PITCH, RATE, RHYTHM AND QUALITY OF THE SPEECH OF CHILDREN HANDICAPPED BY CEREBRAL PALSY

BERNEICE R. RUTHERFORD, M.S.
Dowling School, Minneapolis, Minnesota

I. Introduction: The Problem

The speech of children handicapped by cerebral palsy is frequently referred to in the literature as characteristic spastic speech. More than occasionally this same notation is used in the records of these children. Seldom in even the more recent studies concerning the speech of these children is differentiation made on the basis of the types of cerebral palsies. Since there is an anatomical and physiological differentiation, it seems logical to suppose that there might be a basis for a difference in speech reactions and in therapy.

It is evident at the outset that children handicapped by cerebral palsy have good speech as well as defective speech. The problem, then, is one of finding the differentiating characteristics of the speaking voice rather than defective sound production or stuttering, per se. This study was undertaken in an effort to discover (1) whether the speech of children handicapped by cerebral palsy is different from the speech of other children; (2) if so, what that difference is; (3) whether there is a difference in speech between the children in the two main groups of cerebral palsy; (4) what implication there is for direction of differential speech therapy. The term *speech* is employed in this study with reference to both production of speech and to the attributes of voice. However, it is to the speaking voice that this study is directed.

II. Procedure

Subjects

In this study there are two groups of children handicapped by cerebral palsy, an extrapyramidal or athetoid group of 48 children, and a pyramidal or spastic group of 74 children. There is also a control group of 69 children without cerebral palsy, the total number of subjects investigated being 191. Each group is divided according to speech status into two sub-groups: those with defective speech and with good speech, respectively. The defective speech groups include children whose speech, at the beginning of this study, had been diagnosed by a speech clinician to be stuttering speech, or to involve an articulatory defect with sound substitutions or omissions. The good speech groups include children with no stuttering or articulatory defects. Thus, the designation of speech in this study as *defective* or *good* is based upon the presence or absence of stuttering or sound substitutions or omissions. The presence or absence of

the attributes of speech investigated in the study does not enter into the classification of good or defective speech.

With the exception of ten of the group without cerebral palsy, all of the children were on roll at Dowling School for Handicapped Children during the school years 1939 thru 1943. It seemed desirable to include this group of ten children, selected at random from various parts of the city, who had no physical disabilities. Since these ten had no physical disabilities, they were not enrolled at Dowling. The children used in this study were drawn from all sections of the city of Minneapolis, which tends to minimize the possibility of a selective factor of speech opportunities.

TABLE I. DISTRIBUTION OF GOOD AND DEFECTIVE SPEECH IN THE ATHETOTIC (CPEX), SPASTIC (CPP) AND NON-CEREBRAL PALSY (NON-CP) GROUPS, RESPECTIVELY.

	CPex	CPp	Non-CP
Defective speech	43	48	43
Good speech	5	26	26

Physical diagnoses on all of the children except the ten not enrolled at Dowling were made by the school's consulting orthopedic surgeon, Dr. John F. Pohl. The group designated as without cerebral palsy includes children with various physical disabilities, such as poliomyelitis, cardiac deficiencies and osteomyelitis, as well as those with no physical disabilities. All cases in which the cerebral palsy had been diagnosed as ataxic, or was of known encephalitic origin, were eliminated. Observable bases of differentiation of the two cerebral palsy groups are the bizarre positions and writhing movements of the extrapyramidal or athetoid group and the more rigid, fixed position of the affected parts of the pyramidal or spastic group. Neither the terms *extrapyramidal* and *athetoid*, nor *pyramidal* and *spastic*, are to be thought of as interchangeable. The terms *extrapyramidal* and *pyramidal* designate the anatomical differentiation, and the terms *athetoid* and *spastic* designate the physiological differentiation of the cerebral palsy condition. Up to this point these terms have been used simply to indicate the physiological and anatomical diagnoses and to differentiate the two cerebral palsy groups used in this study. For the purpose of this paper either set of terms could be employed. For the sake of brevity the cerebral palsy extrapyramidal or athetoid groups will be designated as CPex, and the cerebral palsy pyramidal or spastic group as CPp. The group without cerebral palsy will be designated as Non-CP. The speech status will be designated as defective or good.

It should be noted here, perhaps, that not all children handicapped by cerebral palsy have defective speech. It will be observed that in this study the number of children in the CPp group who have good speech is greater than the number in the CPex group. This reflects the situation at Dowling. There are,

however, a considerably larger group of children who have good speech in the Non-CP group. The number in this group in the present study was arbitrarily limited to 26, to match the number in the CPp group.

Chronological age and sex distributions are given in Tables II, III and IV.

TABLE II. RANGE AND MEAN OF CHRONOLOGICAL AGE IN YEARS AND SEX DISTRIBUTION OF THE CHILDREN WITH POOR SPEECH IN EACH OF THE GROUPS STUDIED.

	C.A. Range	C.A. Mean	No. Boys	No. Girls
CPex	4-17	10	24	19
CPp	3-17	6	23	25
Non-CP	4-16	11	14	29

TABLE III. RANGE AND MEAN OF CHRONOLOGICAL AGE IN YEARS AND SEX DISTRIBUTION OF THE CHILDREN WITH GOOD SPEECH IN EACH OF THE GROUPS STUDIED.

	C.A. Range	C.A. Mean	No. Boys	No. Girls
CPex	9-17	12	4	1
CPp	4-16	9	14	12
Non-CP	3-17	9	11	15

TABLE IV. RANGE AND MEAN OF CHRONOLOGICAL AGE IN YEARS AND SEX DISTRIBUTION OF THE TOTAL NUMBER OF CHILDREN IN EACH GROUP STUDIED.

	C.A. Range	C.A. Mean	No. Boys	No. Girls
CPex	4-17	10	28	20
CPp	3-17	7	37	37
Non-CP	3-17	10	25	44

Ratings

The attributes of speech investigated in this study are loudness, pitch, rate, rhythm and quality. Rather coarse scale values are employed in judging pitch, loudness, rate and rhythm because such scale values seem more nearly to approximate terms usually used in describing the speaking voice. The following designations were used:

	1	2	3
Pitch	low	medium	high
Loudness	weak	moderate	loud
Rate	slow	moderate	rapid
Rhythm	smooth	jerky	

Quality was not rated on a scale because the differences noted were assumed to be not of degree but of kind. Quality was classified in each case under one of the following headings: monotony, breathiness, nasality, huskiness and clearness.

Since judgments were to be made of the speaking voice, as natural a speaking situation as could be obtained was desired. Each child individually was asked to give, in sentences, (1) his name and address, (2) the names of his brothers and sisters, and their school grades and (3) to discuss the sport or activity which he most enjoyed. If any one of the judges wished to hear him further, that judge asked questions until he was satisfied with his judgment.

Ratings on 114 of the cases were made by three persons, Miss Gladys McAlister, the principal, Miss Grace Johnson, the occupational therapist, and the author, the speech clinician, all of Dowling School. Ratings on the remaining 77 cases were made by the speech clinician only.

Preliminary to making the ratings on the 114 cases the judges rated 10 cases in joint conference in order to be certain that all three persons were judging on the basis of comparable definitions. Particular attention was given to the differentiation of stuttering speech and the jerky speech heard in some children handicapped by cerebral palsy. The percentage of agreement among the three judges was 81 per cent.

Two separate judgments were made of the 77 cases who were rated only by the speech clinician. The agreement between these two ratings was 97 per cent.

Tests of certain breathing performances were given by the speech clinician to all of the children in this study who were handicapped by cerebral palsy. These tests were designed to indicate the ability each child possessed to pant, gasp, inhale deeply and prolong exhalation. Each child was asked to take a deep breath; to exhale vocally, prolonging the exhalation as long as he could; to take a quick, short breath (gasp); and to pant. If he did not understand the instructions the desired type of performance was demonstrated and he was asked to imitate it. Results of the analysis of the data obtained by these tests are given in Table VII.

Anaylsis of Data

Each of the 114 cases was rated individually by each of the three judges. These judgments were recorded by the individual judges on a form which was set up in an effort to gain uniformity and accuracy as far as possible. That the judgments were subjective is recognized by the writer. After all judgments were made, the ratings by the individual judges were transferred to a master sheet. Tabulations were made from this master sheet. At least two out of the three ratings had to agree in order for a scale value to be assigned to any attribute for any individual.

In the tabulation of the 77 cases rated by the speech clinician alone, a scale value was assigned on the basis of two judgments in the 97 per cent of the cases in which the two judgments were the same. For the remaining three per cent a third rating was made and the scale value was assigned on the basis of two out of the three judgments.

In order to determine whether the differences found with respect to the attributes are real differences or due to chance, chi square was calculated for each attribute. Sub-totals from Table V with corresponding chi square values are given in Table VI.

IV. Results

Results of the study may be discussed with reference to the group differences obtained. A summary of the data is presented in Table V.

In discussing the findings involving the CPex good speech group it should be remembered that the number of cases in this group is small. It is possible, however, that the comparatively small number of children in the CPex group who have good speech may be in itself significant.

TABLE V. Percentages of Cases Classified in Each of the Indicated Speech and Voice Categories in the Athetotic (CPex), Spastic (CPp) and Non-Cerebral Palsy (Non-CP) Groups, with and without Defective Speech.

	Defective Speech			Good Speech		
Attribute	CPex % (N-43)	CPp % (N-48)	Non-CP % (N-43)	CPex % (N-5)	CPp % (N-26)	Non-CP % (N-26)
Loudness						
Weak	12	10	5	0	3	0
Moderate	58	70	81	60	90	100
Loud	30	20	14	40	7	0
Pitch						
Low	21	12	12	20	0	0
Medium	63	73	86	80	100	100
High	16	15	2	0	0	0
Rate						
Slow	51	20	5	0	0	0
Moderate	47	63	88	100	100	100
Rapid	2	17	7	0	0	0
Rhythm						
Smooth	32	80	88	60	93	100
Jerky	68	20	12	40	7	0
Quality						
Monotonous	32	20	5	0	0	4
Breathy	39	10	7	20	3	4
Husky	9	6	7	0	6	12
Nasal	7	12	9	20	3	4
Clear	30	54	72	60	88	80

Non-CP Groups Compared with CP Groups

Though application of chi square to the group differences indicated in Table V shows the differences between the CP and Non-CP groups to be larger than can be accounted for by chance, the trends in each attribute are toward moderate or medium. Table VI shows these relationships.

a. Loudness. Both Non-CP and CP groups in this study tend to have voices of medium loudness, although the trend for the Non-CP groups is more decided. In both groups the number of loud voices exceeds the number of weak voices; in the CPex defective speech group there are more than twice as many loud voices as there are in the Non-CP defective speech group. Variations are greater within the CP group than within the Non-CP group.

b. Pitch. The trend in both Non-CP and CP groups is toward moderate pitch. The number of children in the CP group having higher-than-moderately-pitched voices is almost equal to the number having lower-than-moderately-pitched voices. In the Non-CP group only six cases or 8 per cent vary from the moderate trend, and just one of these was rated

as higher-than-moderately-pitched, while in the CP group approximately 24 per cent vary from a moderate trend.

c. Rate. While the trend of both Non-CP and CP groups is toward the moderate rate, the variation within the CP group is greater than that in the Non-CP group. The trend in the Non-CP group is more definitely toward moderate rate. In this group 93 per cent were judged as having a moderate rate of speech, against 66 per cent of the CP group who were so judged. The CP group is weighted toward slow rate by the comparatively large number in the CPex defective speech group who were rated as having slow speech.

TABLE VI. SIGNIFICANCE OF DIFFERENCES IN LOUDNESS, PITCH, RATE, RHYTHM AND QUALITY.

Attribute	CP	Non-CP	CPp	CPex	Defective Speech	Good Speech
Loudness						
Weak	11	2	6	5	12	1
Moderate	84	61	56	28	93	52
Loud	27	6	12	15	29	4
	$X^2=11.75$		$X^2=5.27$		$X^2=19.21$	
Pitch						
Low	16	5	6	10	20	1
Medium	92	63	21	31	99	56
High	14	1	7	7	15	0
	$X^2=13.85$		$X^2=5.40$		$X^2=22.05$	
Rate						
Slow	32	2	10	22	34	0
Moderate	81	64	56	25	88	57
Rapid	9	3	8	1	12	0
	$X^2=15.496$		$X^2=10.65$		$X^2=26.30$	
Rhythm						
Smooth	79	64	62	17	90	53
Jerky	43	5	12	31	44	4
	$X^2=18.78$		$X^2=17.00$		$X^2=21.45$	
Quality						
Clear	65	52	49	16	70	47
Not clear	57	17	25	32	64	10
	$X^2=11.52$		$X^2=20.63$		$X^2=21.96$	

d. Rhythm. Only five children or 7 per cent of the Non-CP group were rated as having jerky speech. In the CP group there are forty-three children or 35 per cent so rated. The trend in both groups, however, seems to be toward smooth speech, although this trend is much less definite in the CP group (65 per cent) than in the Non-CP group (93 per cent). The CP group is weighted by the number in the CPex group who have jerky speech.

e. Quality. The trend of the Non-CP group is toward speech which is free from the four qualities investigated. In the CP group the number of children who have clear speech, that is, free from the four qualities investigated, approximately equals the number of children who have one or more of these qualities present. Two or more of these qualities are present in nine of the CP group, but in no instance in the Non-CP group are there two or more qualities present.

CPex Groups Compared with CPp Groups

Chi square values for the two CP groups are significantly smaller with reference to loudness, pitch and rate, although

they still indicate that the differences are not due to chance. Trends for the two CP groups are not consistent for all attributes. Table VI shows where the differences lie.

a. Loudness. The trend of both CPex and CPp groups in this study is toward moderate loudness; this trend is stronger in the CPp group. However, there are comparatively more loud voices in the CPex group (30 per cent) than in the CPp group (16 per cent).

b. Pitch. In both CP groups the trend is toward moderate pitch. There are approximately as many children having high-pitched voices in the CPex defective speech group as there are in the CPp group, and slightly more of the CPex defective speech group have low-pitched voices. It should be remembered that the coarseness of the scale used in this study allows opportunities for variations within these high, medium or low ranges.

c. Rate. The rate trends in the two CP groups differ somewhat. The CPex group tends to have slow to moderate rate of speech, with emphasis upon slow. One child has drawling speech. The CPp group trend is more toward a moderate rate. In this group approximately as many children were judged to have slow speech as were judged to have rapid speech.

A study of the test results on movements of the speech articulators of these children indicates that, in general, the CPex group has more difficulty in moving the speech musculature than does the CPp group. These tests include ability to move tip, back, sides and blade of tongue; lips, jaws, palate and throat.

Analysis of the results of the breathing test given to the two CP groups seems to give a partial explanation for this difference also. These tests are briefly described in Section II above. The results given in Table VII are in terms of positive response to a verbal instruction or to a request for imitation of the tester's action.

TABLE VII. DISTRIBUTION OF POSITIVE RESPONSE ON FOUR BREATHING PATTERNS.

	Deep Inspiration				Prolonged Expiration				Panting				Gasping			
	N		%		N		%		N		%		N		%	
	D	G	D	G	D	G	D	G	D	G	D	G	D	G	D	G
Cpex (N Defective Speech: 43, Good Speech: 5)	32	5	74	100	31	5	72	100	30	5	70	100	21	5	70	100
CPp (N Defective Speech: 48, Good Speech: 26)	46	26	96	100	45	26	94	100	35	26	73	100	34	26	71	100

Only one of the CPex group seemed unable to reproduce any of the four patterns; five were unable to reproduce three of the patterns; and eleven were unable to reproduce two of the patterns. Two of the CPp group (both under 6 years of age) failed on all of the four patterns; one child failed to reproduce three of them; and seven failed on two of the patterns. Faulty breathing habits, or lack of control of the breathing apparatus, may be responsible for the slower rate of speech among the CPex defective speech group.

d. Rhythm. The trends in rhythm of the two CP defective speech groups seem to be quite different. Sixty-five per cent of the CPex group have jerky speech, as compared with 16 per cent of the CPp group. The CPex defective speech group shows a trend toward jerky speech, the CPp defective speech group tends toward smooth speech. This interrupted rhythm of the CPex group can be rather readily differentiated from, and should not be confused with, stuttering. Table VII indicates that the CPex group has less ability than the CPp group to control breathing for deep inspirations and prolonged expirations.

e. Quality. There are approximately twice as many children in the CPp group who have clear speech, free from huskiness, monotony, breathiness or nasality, as there are in the CPex group. More breathiness and monotony were found in the CPex defective speech group, and more nasality in the CPp defective speech group; about the same number of cases of husky speech was found in each group. Five were found to have monotonous and breathy speech. One has three qualities present.

Two of CPp defective speech group have more than one of the qualities studied. In these two monotony and nasality are both present.

V. Summary and Conclusions

The anatomical and physiological differentiation between the two types of cerebral palsy, CPex and CPp, would suggest a basis for differences in the speech of children whose physical disabilities were so diagnosed. It would also be supposed that, with motor functioning disturbed, the speech of a child handicapped by cerebral palsy would be different from the speech of a child not so handicapped. This study was undertaken to discover if the speech of these children is different from the speech of children who are not handicapped by cerebral palsy, and also if there is a speech difference between the two cerebral palsy groups.

In this sampling of the speech of children handicapped by cerebral palsy, there is little evidence to indicate that there is a sharply defined type of speech which is peculiarly characteristic of these children. The range of individual differences is wider and there seem to be more extremes in the CP groups than in the Non-CP groups in all the attributes studied. Results of the application of chi square indicate that differences found between Non-CP and CP groups are not due to chance. But, in general, the trends in loudness, pitch, rate, rhythm and quality are the same in the speech of the CP group as in the speech of the Non-CP group.

Within the two CP groups there seems to be a difference in trends which would further indicate that there is no characteristic speech for children handicapped by cerebral palsy in a general sense.

In describing the speech of these children, then, the term *characteristic spastic speech* should be avoided. For, even though there is a difference from the normal in some trends in the CPex (athetotic) group, there is no constant difference in the speech of cerebral palsied children as an entire group.

These data suggest, however, as has been stated, that there is some difference in speech reactions between the two CP groups. They seem to differ in rate and rhythm trends, and in the proportion of voices which are loud, low-pitched, monotonous, or breathy. The CPex (athetotic) group seems to tend toward slower, more jerky speech than the CPp (spastic)

group. In the CPex group there are also more loud voices, more low-pitched voices, and more monotonous or breathy voices.

Examination of the agility of the speech musculature and breathing apparatus seems to point to at least a partial basis for this difference. The attributes and qualities differentiating the two groups—i.e., rate, rhythm, loudness, low pitch, monotony and breathiness—seem to be definitely related to control of the breathing apparatus.

The difference between the two CP groups in breath control and in mobility of the speech musculature suggests that a difference in therapy for CPex and CPp groups is indicated. Differential diagnosis, physiologically and anatomically, is indicated as the basis for initial screening. Further tests of individual abilities within each CP group should disclose individual needs. Differential therapy will then be based upon these needs.

This study seems to indicate that there is no speech which is characteristic of all children handicapped by cerebral palsy; that there is a difference with respect to some speech trends between the two main groups of cerebral palsy; and that a difference in therapy for CPp and CPex groups is indicated.

NEWS AND ANNOUNCEMENTS

(Continued from Page 262)

The University of Syracuse has assigned a building on the campus and near the medical center to provide quarters for the speech laboratory and its remedial activities.

The College of Wooster, Ohio, is establishing a speech clinic. W. C. Craig, who has been chairman of the Department of Speech at Capital University, Columbus, Ohio, for thirteen years, will head the speech correction work at Wooster. His will be the job of planning and developing the speech clinic.

The University of Illinois, Division of Services for Crippled Children, held a conference on speech rehabilitation on March 24, 1944, in Chicago. Miss Jayne Shover was chairman of the morning session. Mr. Lawrence J. Linck, director of the Division of Services for Crippled Children, spoke on the division speech program. Dr. Alan G. Brodie, head of the Department of Orthodontia, University of Illinois, discussed orthodontia. Plastic surgery was the topic of Dr. Louis W. Schultz, assistant professor of oral surgery, University of Illinois.

Dr. Severina Nelson presided over the afternoon session. Dr. Clarence T. Simon presented a paper on clinical equipment. Mrs. Frances Gaines, Children's Memorial Hospital, discussed examination and case history. Various effective techniques and exercises were outlined by Dr. Mildred Berry, Rockford College, Dr. Doris Twitchell, Northern Illinois State Teachers College, and Dr. Florence Henderson, University of Illinois. The matter of social adjustment was the subject of a talk by Dr. Lincoln D. Holmes, Illinois State Normal University. The field of special education was discussed by Mr. Roy Graham, assistant superintendent of public instruction, Springfield, Illinois.

The Institute of Logopedics of the Municipal University of Wichita is emphasizing work with the hard-of-hearing and the rehabilitation of men with aphasia and facial and laryngeal injuries. There are now available a number of scholarships for graduate work at the Institute. The Institute will be glad to hear of any prospective student who wishes to take advantage of the clinical opportunities that exist.

In cooperation with certain army hospitals and rehabilitation programs, the School of Speech of the University of Denver is conducting an experiment in combining the work of psychodramatics, psychospeech methods and general semantics for the assistance of so-called psychoneurotics. The experiment is being conducted by Dr. Elwood Murray, director of the

(Continued on Page 276)

AUDITORY MEMORY SPAN FOR MEANINGLESS SYLLABLES

HELEN HULICK BEEBE
Easton, Pennsylvania

I.

According to a study made by Weiss (4), auditory memory span for a series of meaningless syllables is correlated with chronological age. Normally a child of three years repeated three syllables, one of four years four syllables and so on, progressing to about the ninth year. Both Weiss (4) and Froeschels (1) found that in cases of paragrammatism (this term is used in preference to agrammatism which implies speaking *without* grammar) auditory memory span fell below this level. Froeschels gives the following table of averages from a group of paragrammatic boys and girls:

Age	No. of Syllables
6	3.4
6.5	3.1
7	3.6
7.5	4.2
8	3.8
8.5	4.1
9	5.0

Both Weiss and Froeschels were working with German-speaking children. It is known that German grammar is largely a matter of syllable arrangement. For example, *geben*=give, *gegeben*=have given; thus a German-speaking child must give acoustic attention to the selection of syllables if he is to acquire skill in grammar and syntax.

The importance of auditory memory span is emphasized by West, Kennedy and Carr (5, 162-163). They state that a child of three should be able to recall two sounds, a child of eight an average of two and one-half sounds, and a pubescent child an average well above three sounds, or as many as an adult. These writers think that marked deviations from norms should be regarded as having serious etiological possibilities. Although mention is made of the importance of using sound combinations without conventionalized meanings, it is not clear whether *sounds* designate syllables or single phonetic elements. For example, would three sounds be *golabee* or *tob*?

S. D. Robbins (3) studied the relation between short memory span and disorders of speech. He found that auditory memory span for logical sentences is considerably longer than for digits, consonants, and nonsense syllables. He gives the average auditory memory span for digits as:

Age	No. of Digits
3	3
4	4
7	5
10	6
14	7
18	8

In testing cases of speech disability for digit auditory memory span Robbins came to the conclusion that while a short span seems to be an etiological factor in dyslalia, this does not appear to hold in cases of stammering and cluttering. Robbins refers to the fact that A. S. Clark found no correlation between auditory memory span and intelligence.

R. W. Metraux (2) reviews the controversial phases found in the literature on auditory memory span in her introduction to a study made to find the significance of auditory memory span in relation to age, sex, and intelligence. Metraux used a modification of Anderson's test, administered with phonograph records. The subjects were tested for memory of vowel and consonant sounds separately. The results of the tests showed differences in auditory memory span among the different age groups (5 to 12 years), the span increasing with age although the peak for vowels is reached at age 10 while the peak for consonants is reached at age 12. The differences between auditory memory span for boys and girls and the relation between auditory memory span and mental age were not considered significant according to this study.

II.

The present writer was interested in comparing English-speaking children with German-speaking children in their ability to repeat a series of meaningless syllables. For this purpose a group of normal speaking public school children were used. The group numbered 207, including 86 boys and 121 girls, ranging in age from 4 years to 8 years 5 months.

The test and the scoring were less elaborate than that used by Metraux but followed as closely as possible the procedure used by Weiss and Froeschels. The following procedure was used: The writer, acting as examiner, explained to each child that he was to turn his back and repeat some sounds which she was going to say such as, *goulabi.*[1] Care was taken to control as far as possible the factors of inflection, rhythm, etc. The series of syllables were given at speech tempo without interruption. The same set of series was used for each child starting with the three syllables, *padigou.*[1] If he repeated these correctly he was given a new series of four syllables and so on until his capacity was reached. The number in the series immediately preceding the first failure was used as the child's score. (In four cases who failed to repeat three syllables it was necessary to offer two.) The results are given in Table I.

Most of the 6-, 7,- and 8-year-old groups had had intelligence tests (revised Stanford Binet). Table III shows the average intelligence quotients for these three age groups as they are related to the number of syllables scored. For example, the average I.Q. for all 6-year-old children who could repeat three syllables is 105, for those repeating four syllables, 119, etc.

[1]International Phonetic Alphabet.

III.

According to these data *auditory memory span for meaningless syllables* increases with chronological age, but not consistently with each change in age level, for English-speaking children. As against these averages those given by West, Kennedy and Carr seem low. The average auditory memory span values shown in Table I fall below those for German-speaking children. The latter fact may be accounted for if we consider the importance of syllable arrangement in the construction of the German language.

TABLE I. DISTRIBUTION AND MEANS OF AUDITORY MEMORY SPAN SCORES IN TERMS OF NUMBER OF MEANINGLESS SYLLABLES CORRECTLY REPEATED.

	Chronological Age*				
No. of Syllables	4 (N=11)	5 (N=47)	6 (N=31)	7 (N=60)	8 (N=58)
2		2	2		
3	2	11	4	8	3
4	7	18	13	20	23
5	2	6	8	26	22
6		4	4	6	10
Average No. of Syllables	4	3.8	4.3	4.3	4.6

*Age 4 includes all children between 3 years 6 months and 4 years 5 months, age 5 all children between 4 years 6 months and 5 years 5 months, etc.

There is no significant difference between the auditory memory span of boys and girls, as shown in Table II. This is in agreement with the findings of Metraux and others.

TABLE II. MEANS OF AUDITORY MEMORY SPAN FOR BOYS AND GIRLS OF EACH AGE GROUP.

	Average No. of Syllables	
Age	Boys	Girls
4	4.3	4
5	4	3.8
6	4.8	4.1
7	4.3	4.6
8	4.5	4.7

TABLE III. RELATIONSHIP BETWEEN AUDITORY MEMORY SPAN AND INTELLIGENCE QUOTIENTS FOR AGE GROUPS 6–8 YEARS.

No. of Syllables	Average Intelligence Quotients		
	Age 6	Age 7	Age 8
3	105	102	97
4	119	107	112
5	119	113	103
6	117	115	107
Range of I Q	99–143	79–131	84–148

The small number of subjects and the limited age groups do not permit any definite conclusions about the relationship of intelligence to auditory memory span for meaningless syllables. The differences shown in Table III are not great enough to be significant but there is enough variation to indicate that further research on this question would be justified.

REFERENCES

1. Froeschels, Emil. Zur Frage des kindlichen Paragrammatisms. *Weiner med. Wchnschr.*, 1930, No. 35.
2. Metraux, Ruth Watt. Auditory memory span for speech sounds: norms for children. *J. Speech Disorders*, 1944, 9, 31-38.
3. Robbins, S. D. The relations between short auditory memory span disability and disorders of speech. *Laryngoscope*, 1935, 45, 545-553.
4. Weiss, D. Testuntersuchugen an normalen und sprachgestörten Kindern. *Weiner med. Wchnschr.*, 1930, No. 32.
5. West, Robert W., Kennedy, Lou, and Carr, Anna. The rehabilitation of speech. New York: Harpers, 1937.

NEWS AND ANNOUNCEMENTS

(Continued from Page 272)

School of Speech, and Dr. Ernest Henrikson, director of the speech clinic of the University of Denver.

Dr. Henrikson and Mrs. Edna Hill Young are presenting a special training clinic and laboratory in the correction of war-induced speech disorders. The work is being given both at rehabilitation centers for the workers with returned soldiers and at the University for students preparing for careers in the field of rehabilitation.

The ninth annual assembly of the International College of Surgeons will be held October 3-5, 1944, at the Benjamin Franklin Hotel in Philadelphia, Pa. The program will be devoted to war, rehabilitation and civilian surgery. The medical profession is invited to attend the assembly and its sessions.

The following statement was released June 10, 1944, by the Federal Security Agency of the U. S. Office of Education:

Lester A. Kirkendall has been temporarily assigned within the Federal Security Agency, from the U. S. Public Health Service, to serve as senior specialist in health education in the Division of Physical Education and Health Activities in the U. S. Office of Education. He will serve schools and colleges in the broad area variously designated as social hygiene, "human relations," etc. The need of this service is attested by the many requests for aid received from state and local school officers, by resolution of the National Conference for Cooperation in School Health Education, which includes most of the public and private health agencies that have for many generations served our schools, and by the conviction of the Surgeon General of the U. S. Public Health Service that appropriate school programs are needed to develop a sound basic approach in this area of human life that has such far-reaching implications for America's health.

Dr. Kirkendall's services are available to school systems, to teacher-training institutions, to workshops, and to other agencies and organizations devoted to the education of American youth.

At their spring meeting, the Minnesota Speech Clinicians Association elected the following officers: president, Katherine Thorn, University of Minnesota Speech Clinic; vice-president, Theo. Olson, Austin, Minnesota; secretary-treasurer, Florence Haavik, St. Louis Park, Minnesota. Plans were made for the Minnesota Speech Clinicians to meet during the Minnesota Educational Association state-wide meeting in October, 1944.

Shady Trails, the national speech improvement camp for boys, had another successful summer. They had a full quota of fifty-five boys and had to turn down numerous applications. J. N. Clancy is the director of the camp and had a staff of nineteen. The staff included teachers of speech correction, physical directors and administrators.

Dr. Ralph R. Sokolowsky, otolaryngologist and phoniatrist, Fellow of ASCA, died on June 25, 1944, following a cerebral hemorrhage.

Dr. Sokolowsky was born in 1876 in Kovno (Lithuania) Russia, attended the Royal Prussian Gymnasium in Lyck (East Prussia), and received the degree of Doctor of Medicine. During subsequent study he was connected with several of the leading otolaryngological clinics in Berlin and Konigsberg. From 1923 to 1934 he served as Professor Extraordinarius at the University of Konigsberg. His private patients in Konigsberg included many prominent singers and speakers of Europe. From February, 1940, until his death he was associated with the Department of Speech of Capital University as speech pathologist and lecturer in speech and voice correction.

His international publications in the field of corrective speech bear testimony to Dr. Sokolowsky's careful work. He published studies in a number of languages, and was intimately acquainted with the literature of speech and voice correction of many countries. Two works were awaiting publication at the time of his death, a book, written in collaboration with the undersigned, and an article concerning war aphonia, which appears in this issue of the *Journal*.

Those of us who were associated with Dr. Sokolowsky feel, in the passing of this kindly, retiring, modest man, a great sense of personal loss. The entire field of speech science has lost a scholar who was most exacting in research, most astute in practice, and most constant in enthusiasm. *(W. C. Craig.)*

PERSONALS

Miss Bette Bartell and Miss Patricia Baldridge are now serving as assistants in the audiometric and hearing aid program of the Department of Otolaryngology at the University of Iowa. Miss Bartell was a speech correction teacher in the Rochester, Minnesota, public schools last year.

Mrs. Edna Hill Young of the University of Denver was awarded the honorary degree of Doctor of Pedagogy by Hastings College at the June commencement. The degree was awarded for her contributions in originating and developing the moto-kinesthetic methods of speech correction and for the contributions which these methods have made in work with children with delayed and defective speech.

Dr. Raymond Carhart has been granted a leave of absence from Northwestern University in order that he might accept the post of psycho-acoustic physicist at Deshon General Hospital, Butler, Pennsylvania. Deshon is one of three army hospitals with units engaged in rehabilitation of deafened military personnel.

Stanley Ainsworth was a member of the Speech Re-education Department of Northwestern University this past summer, teaching courses in speech pathology and phonetics. Mr. Ainsworth is clinic supervisor of speech therapy at Indiana State Teachers College.

Miss Laila Larsen is now at Deshon General Hospital, Butler, Pennsylvania, in charge of building up the acoustic training department. It is her responsibility to see that deafened military personnel learn to use their hearing aids to the utmost advantage.

James C. Kelly was commissioned in the Navy in June and reported to the Bureau of Medicine and Surgery in Washington, D. C. He was a member of the Speech Department of Indiana State Teachers College.

Dr. Gordon Peterson is on leave of absence from Northwestern University and has joined the staff of the psycho-acoustic laboratory at Harvard University.

Genevieve Arnold, a teacher of speech at the University of Hawaii, is now doing graduate work in child welfare and speech pathology at the University of Minnesota.

During the summer just past, the following served as assistants on the staff of the University of Iowa speech clinic: Margaret Eckhart, Rochester, Minnesota; Margaret Kennedy, Davenport, Iowa; Hildred Schuell, South Bend, Indiana; Gladys Simpson, Ironwood, Michigan; and Eloise Tupper and Joyce Hostettler, graduate students at the University.

Miss Jacqueline Keaster was a visiting staff member of the University of Iowa speech clinic, offering courses in lip reading and speech correction for the hard of hearing. She is now on duty at Borden General Hospital, Chickasha, Oklahoma, where she is doing rehabilitation work with acoustically injured soldiers.

Lt. (jg) Frances Patton, Women's Reserve USN, is now stationed at Philadelphia Naval Hospital to do speech correction work. She is on leave for the duration from the speech clinic of the University of Illinois.

Miss Miriam D. Pauls, of the special education clinics, Indiana State Teachers College, and News and Announcements editor for the *Journal*, has joined the WAVES and is now in training at Smith College, Northampton, Massachusetts.

Professor Grant Fairbanks, Department of Speech, University of Iowa, is on leave from the university and on duty as acoustic physicist at the Borden General Hospital, Chickasha, Oklahoma. His work is concerned with certain aspects of rehabilitation of the war-deafened.

Miss Lorraine Anson resigned in July from her position on the staff of the Otological clinic at the University of Iowa to become the bride of Howard Dahl, director of radio station WKBH. They will reside at 2210 King Street, LaCrosse, Wisconsin.

Professor Claude E. Kantner, director of the speech clinic at Louisiana State University, is leaving in September to assume the position of speech correctionist in the Crippled Children's Division of the University of Oregon Medical School.

WAR NOTES

HERBERT KOEPP-BAKER, *Editor*
The Pennsylvania State College

Borden General Hospital at Chickasha, Oklahoma, Hoff General Hospital at Santa Barbara, California, and Deshon General Hospital at Butler, Pennsylvania, have recently added several new members to their staffs serving the deafened, and two or three persons to their speech correction services.

The Rehabilitation Committee of the Association is maintaining close contact with the program for the deafened, and for those suffering from speech disorders, that is now being formulated by the Navy for its hospital in Philadelphia. The Navy has thoroughly studied the service outline, equipment and personnel of the Army Hospital at Deshon. Although the Navy rehabilitation services will probably assume a somewhat different form from those of the Army, provision is being made for the reeducation of all types of speech-handicapped persons.

The National Defense Research Council is assisting the authorities of the Philadelphia Naval Hospital in the design of its new hearing and speech building. They are also receiving assistance from the ASCA Rehabilitation Committee and the Coordinator in the selection, training and organization of its personnel. It is the policy of the Navy to use only persons in the Naval services in rehabilitation work.

A group of WAVES is soon to begin a program of training in speech-reading instruction in the Philadelphia Unit.

Administrators in charge of rehabilitation for the Veterans' Facilities will probably also adapt their program of care of speech and hearing cases to their own conditions, but will draw heavily for suggestions upon the Navy's experience with its Hearing and Speech Unit at Philadelphia.

Recent advices from the medical rehabilitation authorities of the Army and Navy indicate that although some increase in the number of war-induced speech disorders may be expected next year, this problem will not assume anticipated proportions. Hearing-loss cases now represent a more rapidly growing rehabilitation problem. It can now be said, however, that even in this area the original estimates were too high.

Therefore, the Rehabilitation Committee of the Association urges the membership of the Association to direct major attention to the problem of speech rehabilitation on the local level. Since it appears unlikely that large numbers of speech correctionists will be employed by the Army, Navy, or Veterans' Administration in hospital situations, it would seem to

the best interest of our profession to study local rehabilitation organizations and to identify the service of speech correction with them.

Large numbers of veterans are being discharged into civilian life daily. In the process of vocational rehabilitation speech correction will find a useful place. This will, however, depend largely upon the initiative of speech correctionists in their local communities. The Committee urges speech correctionists to study all educational, clinical and social agencies in their communities, and to offer at once to serve speech- and hearing-handicapped persons.

Present information shows that it is probable that large numbers of either active servicemen or veterans will not be gathered in centers for speech reeducation. Those in charge of service programs for the Army and Navy insist that because of the scattered incidence of these disorders, it will be necessary for those servicemen to be reeducated in their local communities where speech correctionists, now engaged in education or in specialized clinical services, are available.

ABSTRACTS OF CURRENT ARTICLES

MARY HUBER, *Editor*
Brooklyn College

Butt, Arthur J. The veteran returns. *Volta Review*, 1944, 46, 281-300.

A young medical officer, wounded in combat, discusses from the point of view of both patient and doctor the problem of those suddenly made hard of hearing. Such a handicap, resulting suddenly, as from battle explosions, is felt more intensely by the individual than is a gradual loss in hearing acuity. The general health of the patient, his ability to use language, and his ease of adaptability, determine the extent to which he can profit from rehabilitation. It is important that corrective measures, those relating to ear pathology and those concerned with retraining, be undertaken promptly. The latter should be started before the patient loses his memory of sound. The author mentions courses in lip reading, speech correction and speech prophylaxis at rehabilitation centers as part of the treatment given him on his return to the United States. He also mentions that assistance in finding suitable employment is given to the hard of hearing by the Veterans Administration Rehabilitation Bureau, the United States Employment Service, the National Clearing House Committee and the American Red Cross. *(Abstracted by Wynett Barnett, Brooklyn College.)*

Drager, Glenn A. Nerve fibers within the cranio-pharyngeal canal. *The Anatomical Record*, 1944, 88, 235-244.

Drager, working under the auspices of the Institute of Anatomy at the University of Minnesota, investigated the origin, composition and distribution of nerve fibers within the cranio-pharyngeal canal in the sphenoid bone. Material for the study was obtained from eight adult cats.

It was found that the majority of the fibers was autonomic in origin, coming from the internal carotid plexus. Nerve fibers and blood vessels were found to be very closely associated within the canal. A vasomotor effect is posited as the result of activity of these nerve fibers. Drager

writes, ". . . in experiments involving cranial sympathectomy, the possibility of autonomic fibers entering the cranium via the cranio-pharyngeal canal should be kept in mind." Although the cranio-pharyngeal canal is usually closed during gestation in the human, experimental evidence indicates that the canal is open in ten per cent of newborn humans and remains open in three per cent of adult humans.

In addition to its function in housing nerve fibers and blood vessels, the cranio-pharyngeal canal and its contents may be significant in certain endocrinopathies because of its proximity to the hypophysis. *(Abstracted by Jeanette Anderson, Purdue.)*

Giblin, N., and Alley, A. Studies in skull growth. Coronal suture fixation. *The Anatomical Record*, 1944, 88, 143-153.

The findings of Giblin and Alley of the Department of Physiology, McGill University, Montreal, are interesting to, and may be significant in their application for, the speech pathologist who is concerned with atypical development of the brain as an etiological factor in certain cases of retarded, absent, or interrupted linguistic activity.

As a result of experiments conducted on puppies, Giblin and Alley conclude that growth of the skull at any single point would be impossible if the rest of the skull were rigid. They also posit that disturbance of growth at one point affects the rate and direction of growth in other areas. The article is illustrated with seven interesting plates. *(Abstracted by Jeanette Anderson, Purdue.)*

Gilliland, A. R., and Shotwell, Anna M. A preliminary scale for the measurement of the mentality of infants. *Child Development*, 1943, 14, 167-177.

Because the tests constructed by earlier investigators generally start not earlier than three months these authors began the construction of a new battery of tests for use with younger infants. A battery of thirty-two tests was assembled. About one-third of these were new, another one-third were adaptations of other tests and the rest were taken directly as used by previous investigators.

The evidence thus far collected strongly suggests that it will be possible to construct a test that will possess considerable validity and reliability and will have accomplished one more step in the construction of accurate measures of mental development for that period of life in which its rate is relatively the most rapid. *(Abstracted by Arthur Secord, Brooklyn College.)*

Henderson, J. L. Infection in the newborn. *Edinburgh Medical Journal*, 1943, 50, 535-553.

In the three years, 1940-1942, 382 of the 8,075 infants born at the Simpson Maternity Hospital, Edinburgh, died within one month of birth. The cause of death was assigned on the basis of laboratory and clinical findings in 369 cases.

In 28.8% of the cases, death was due to asphyxia; 22.0% of the deaths were caused by infection; intracranial hemorrhage was responsible for 15.4% of the deaths; in 13.8% of the deaths, developmental malformation was the causative factor; 12.5% of the deaths were attributed to prematurity. Seventy-one per cent of the total number of deaths occurred within the first week after birth. In the first three days after birth, asphyxia, intracranial hemorrhage and prematurity accounted for a majority of the deaths.

It is significant for the speech pathologist to note these five major causes of infant death. If they do not cause death, it is reasonable to assume that they may be held responsible for retardation in development later in the child's life; speech may be one of the activities affected. *(Abstracted by Jeanette Anderson, Purdue.)*

Johnson, Wendell. Ten children you should know. *National Parent-Teacher*, 1944, 38, 10-12.

Of the ten speech defectives in an average group of 100 American youngsters, four or five will be found to have defects of more serious and complex types. One or two may not speak properly because of hearing losses. The remainder may include those who stutter or suffer from malocclusion, cleft palate, with or without hare lip, deficient motor coordination or low mental ability, or are simply retarded because of faulty training or personality maladjustment. The latter are school children and are being influenced by school teachers. Public school superintendents should be familiar with these facts and every time one interviews a candidate for a teaching position, regardless of the subjects she proposes to teach, he should ask her what she knows about handling children with speech defects in the classroom. If her answer is weak, he has some obligation to make properly pointed inquiries of the college or university in which she was trained. The salary of a speech correction teacher, who can give personal attention to serious cases and direct the classroom teacher in helping other children less seriously handicapped, is one of the finest investments a school board can make.

It is the obligation of parents and teachers to help a child develop his assets and to be aware of them; and at the same time to feel that he need not apologize for his liabilities. This is particularly true of the child with a speech defect. He, too, is a member of a democracy. *(Abstracted by Wynett Barnett, Brooklyn College.)*

Langer, Susanne K. The lord of creation. *Fortune*, 1944, 29, 1-127.

Mrs. Langer, a philosopher, logician and educator who taught for many years at Radcliffe College, examines man's behavior in an attempt to uncover reasons for it. Her investigations lead her to scrutinize his linguistic behavior.

Man's ability to perceive not only actualities but meanings as well is the principal factor differentiating him from other animals. Because he lives not only through sense but also by means of symbols, man has become the master of earth and all its progeny. Not only can man use symbols to hold ideas after reality has passed away; he can associate and re-associate symbols in his mind to formulate things and events that do not exist in the real world. This power is called imagination. Man can also symbolize an idea partially, letting the rest of it go; this power is known as abstraction. Of these abilities, Mrs. Langer writes, "The combined effect of these two powers is inestimable. They are the roots of his supreme talent, the gift of reason."

In evaluating man's use of his powers derived from his ability to manipulate symbols, Mrs. Langer concludes that man is essentially unrealistic. The animal mind is likened to a telephone exchange; the human mind is compared to a great projector or transformer. Ideas, according to Mrs. Langer, "are the transformations of actual things. They are, in fact, *symbols* of reality, not pieces of it."

Careful to distinguish between sign and symbol in her discussion, Mrs. Langer defines these two terms. "A sign is anything that announces the existence or the imminence of some event, the presence of a thing or of a person, or a change in a state of affairs . . . In every case a sign is closely bound up with something noted or to be expected in experience. It is always a part of the situation to which it refers, though the *reference* may be remote in space and time. In so far as we are led to note or expect the signified event we are making correct use of a sign. This is the essence of rational behavior, which animals show in varying degrees. It is entirely realistic . . ."

If man were entirely a sign-using animal, he would perfect the same sort of biological cycle accomplished by animal generations; but he is the only symbol-using animal and so lives differently. His mode of living differs from that of other animals as symbols differ from signs.

"A symbol differs from a sign in that it does not announce the presence of the object, the being, condition or whatnot, which is its meaning, but merely *brings this thing to mind* . . . The difference between a sign and a symbol is, in brief, that a sign causes us to think or act *in face* of the thing signified, whereas a symbol causes us to think *about* the thing symbolized."

Her explanation of the origin of language is not so complete. She says "Conception, not social control, is its first and foremost benefit." In her eagerness to consider the individual and his behavior, Mrs. Langer tends to look upon language as an end in itself rather than as a tool or a means of manipulation. She seems to contradict herself when she maintains that a baby uses a word before he associates it with an object and, at the same time, that language must be denotative from the beginning.

She posits that men are unrealistic because they can play with ideas; that this ability inclines men "to neglect the all-important interpretation of signs for a rapt contemplation of symbols."

From this generalization, Mrs. Langer reasons that the gift of symbolism or reason is at the same time man's peculiar strength and weakness, for it brings to him alone of the animal world the danger of purely mental aberrations, of losing touch with reality. Confusion of symbols—"symbolic forms that have acquired a false factual status"—subjects the user of symbols to insanity, according to this writer. As she phrases it, "The threat of madness is the price of reason."

In concluding this discussion, Mrs. Langer postulates that we are living in no definite culture but rather in a period between an exhausted civilization and an age yet unborn. Our present language is incapable of formulating and expressing these new conceptions; therefore, our age seems to be one of unreason and madness while it is in reality one of transition and renaissance. *(Abstracted by Jeanette Anderson, Purdue.)*

Strommen, Einar. Statistical trends among hearing aid users. *J. Acoust. Soc. Amer.*, 1944, 15, 211-222.

Each of 10,000 buyers of the Model A-55 Acousticon vacuum-tube hearing aid in the first year following its release in March, 1942, furnished the following information: Age and sex; whether a previous or a new hearing aid user; cause of deafness; whether other members of the family are hard of hearing; whether the person could hear better in loud noise than in quiet conditions; whether head noises were experienced in association with the deafness; stage of deafness (U. S. Public Health Service definitions); B-battery voltage selected; pattern of accentuated amplification selected. These data are summarized and compared in thirteen tables. Comparisons with National Health Survey data are made.

Women outnumbered men in this sample by 5643 to 4357. About 50% of the total fell in the age range from 45 to 64. Over 60% had previously used hearing aids; it is pointed out that these cases need fundamental reeducation of hearing and speech habits. A much higher proportion of men (74.2%) than women (47.6%) required high-frequency accentuation. The question, "Do you hear better in a noisy place?" is significant in segregating persons with conductive deafness; the question on head noises is not. "Catarrhal conditions" are more than twice as numerous as systemic diseases among the causes of deafness. About 20% of these hearing aid buyers come from families in which there are one or more other persons who are hard of hearing. Only a small proportion acknowledge having sought medical treatment for their deafness. *(Abstracted by William J. Temple, Brooklyn College.)*

Tucker, R. Whitney. Notes on the Philadelphia dialect. *Amer. Speech*, 1944, 19, 37-42.

Tucker (Pennsylvania Military College) discusses the "Philadelphia and neighborhood" dialect from the points of view of vocabulary and of

phonetic characteristics. After a phonetic analysis of vowel differences he asks two questions and leaves both unanswered: Do Philadelphia and Cockney vowel sounds foreshadow a future generalized development in English speech similar to that started about 400 B. C., when Boeotian Greek developed a set of values for the Greek vowels which appeared a few centuries later in all the other dialects? If so, could this be considered a continuation of the series of vowel shifts that have characterized Modern English since the end of the fifteenth century? *(Abstracted by Jeanette Anderson, Purdue.)*

Vann, Herbert M., and Miller, Roland E. A case of pseudo-monoventricular heart terminating in brain abscesses. *The Anatomical Record*, 1944, 88, 155-160.

Vann and Miller report this case from the Department of Anatomy, Bowman Gray School of Medicine, Winston-Salem, North Carolina. At autopsy an eight-year-old boy was found to have bilateral frontal lobe abscesses and congenital absence of the inter-ventricular septum of the heart. There was no indication of endocarditis and no lung abscesses. "It may be assumed, therefore," write the authors, "that the path of infection might have led directly through the heart to the cerebral lobes." Twenty-three cases of monoventricularism with cerebral abscess as the terminal illness have been reported in the literature prior to this one. One of the patients was over 50 years of age at the time of death, and several were in their middle or late thirties. This report is of interest to the speech pathologist because of cases in which aphasic manifestations may be caused by cerebral abscess. *(Abstracted by Jeanette Anderson, Purdue.)*

Will, Nell. The personality development of a stuttering boy. The Quarterly *Journal of Speech*, 1944, **30**, 88-95. Reprinted in *Etc.: A Review of General Semantics*, 1944, **1**, 165-173.

This is a very comprehensive report of a thirteen-year-old boy, who had stuttered for seven years; it describes the procedures and the effects of a six-month program of retraining. The treatment consisted of approaching the child from two angles: from "within himself" and from his environment. The child was first made to feel that he was a worthy individual by looking up to him and by making him feel that what he had to say was important. Secondly, the child was given certain tools with which to attack his fears. These tools consisted of (1) semantic relaxation, (2) improved word-fact relationships, (3) changing of the child's attitudes toward himself as a personality, and (4) changing his attitudes toward his speech. He was also trained in voluntary performance of non-fluency in speech.

In summarizing the results of this program of retraining Mrs. Will reports that the child seems to be almost as fluent as the average normal speaker, has confidence in himself as a speaker, and has made his first satisfactory school adjustment since he started to school seven years ago. *(M. H.)*

Yoakam, Doris G. Speech games for children. *The Quarterly Journal of Speech*, 1944, **30**, 85-87.

Two cautions must always be kept in mind in using common games for speech improvement lessons. First the goal must continually be emphasized. The purpose is to master the speech sound, not just win the game. The second caution is that the game must be kept simple, and the materials allowed to become complicated only in accord with the ability of the child. The author presents some valuable suggestions for using parlor games for speech correction and stresses the need for variety in children's speech lessons which can be made almost limitless by a judicious application of inexpensive parlor games like checkers, dominoes, lotto, and pick-up-sticks. *(M. H.)*

BOOK REVIEWS

ERNEST H. HENRIKSON, *Editor*
University of Denver

WHYTE, JOHN. American Words and their Ways—Especially for German Americans. New York: The Viking Press, 1943. Pp. xvi + 184. $2.50.

The author and his purpose

The author indicates clearly, not only in the title of the book but also in his remarks in the preface, that he is writing specifically for a certain group, that is, for German Americans. In general the book is designed to aid the members of this group in acquiring the ability to speak and write colloquial American English and in adapting themselves to life in America. This purpose is evidenced by the three main subdivisions: Speaking and Writing Colloquial American English, Social Forms and Social Customs in America, and Differences in National Characteristics and Temperament.

Mr. Whyte was himself born and reared among German Americans in Watertown, Wisconsin. He has, in the words of the fly-leaf, "lived a number of years in Germany and studied in the Universities of Leipzig and Berlin." He is currently chairman of the German Department at Brooklyn College. The author in his acknowledgements gives credit to a number of distinguished scholars, particularly in the fields of English and German, for suggestions, criticisms and aid in the preparation of the manuscript.

The contents of the book

There are in all 40 chapters filling 157 pages, an appendix of 24 pages, and an index. The chapters are too numerous to list in detail, but a general summary will indicate the scope of the book. The section on speaking and writing devotes 20 pages to the problem of pronunciation. The author first discusses "standard pronunciation," following the usual pattern of dividing American speech into Eastern, Southern, and General American. He advises the German American to adopt the "cultured speech" of his surroundings, but excepts those living in the South because "the chances are wholly against his ever completely appropriating Southern speech." Mr. Whyte then classifies the speech of 12 well-known radio speakers and devotes 10 pages to a comparison of English and German speech sounds and speech melodies.

The remainder of this section, 83 pages, is given over largely to the writing of English including grammatical problems, slang, a comparison of American and British English, punctuation, syllabification and ending with 100 common errors with corrections. This portion of the book is characterized by its emphasis upon colloquial English and good usage rather than formal English and rules, and by frequent comparisons of the two languages with many examples in German.

The second section on social forms and customs in America presents an abridged summary of what is commonly called "etiquette," with liberal examples comparing and contrasting customs in the two countries. Good form is discussed in relation to greetings and leave-takings, table manners, social calls and invitations, letter writing, tipping, etc.

In part three the author, after cautiously pointing out the dangers of generalization, attempts to discuss some of the main general differences in national characteristics and temperaments between the native born American and the German immigrant. The appendix includes a synopsis of the verb *to build*, a list of prepositions, another of clichés, a third on the pronunciation and spelling of names and foreign words, a short discussion of American and German education, and several summaries of related reading material.

The reviewer's evaluation of the book

First of all it seems clear that this book will be most useful to the German American who is reasonably literate in both languages. The Eng-

lish vocabulary used is that of a well-educated American and many of the most useful comparisons and illustrations are in German. A knowledge of German grammar seems to be assumed. An uneducated German immigrant, or a second-generation German who did not know the language, or an individual with a limited vocabulary in English would be seriously handicapped in getting the full benefit of the book.

Secondly, the reviewer believes that the section on pronunciation is quite inadequate. It contains some good general advice and the discussion of the sounds of the two languages is probably as accurate as possible in a very short and gross description. Nevertheless it is difficult to see how anyone with a real German dialect could profit very greatly from this portion of the book.

In contrast the material on written English and on general problems of usage is detailed and well put. Given the willingness to study and practice, it should be very helpful. A few "purists" may be offended at the consistent following of the rules of usage rather than those of the grammar books, but this procedure will probably meet with general approval.

The last two sections likewise are very helpful and common-sense discussions of many items that should help the German American in adjusting himself to life in this country. Perhaps the greatest merit of the book lies in the fact that it is written for a particular group. This has enabled the author to use many specific illustrations and comparisons and to highlight special problems. The reviewer intends to make this book required reading for any German accent case, within the limitations mentioned earlier, that may come to him in the future, but he does not expect it to lessen materially the amount of training needed in spoken English. He also believes that it could be read with profit by those who work with such cases.

CLAUDE E. KANTNER

Louisiana State University

GOULD, GEORGE M., M.A., M.D. Gould's Pocket Pronouncing Medical Dictionary. Philadelphia: The Blakiston Co., 1943 Printing. Eleventh Edition Revised. $2.00.

The literature of speech correction must of necessity draw on medical terminology. Further, the speech pathologist is vitally interested in many significant writings relating to his field done by medical people. For these and, no doubt, many other reasons, this new edition of Gould's *Pocket Pronouncing Medical Dictionary* should prove of real value to workers in speech rehabilitation. The book is compact and well-bound. In addition to the pronunciation and a concise definition of each of over 40,000 terms, it contains tables of arteries, bones, and muscles and much additional valuable material.

E. H. HENRIKSON

University of Denver

HELTMAN, HARRY JOSEPH. First Aids for Stutterers. Boston: Expression Company, 1943. Pp. 195, with 60 pp. of exercises. $3.00.

The author's purpose in this publication is clearly revealed on the title page: "Practical Steps for Prevention and Treatment of Stuttering. Tested means for use of parents and teachers in the prevention and correction of stuttering and for the adult who wishes to achieve permanent recovery."

It is refreshing to one who has watched the development of the field of rehabilitation of stuttering to see in this book the flowering of a real eclecticism. Just as medicine has progressed from a divergence of schools such as allopathy, homeopathy, and naturopathy to the modern eclectic school, so the treatment of stuttering has emerged from "systems" to

individualized and selective practice based upon the contributions of all who have done good work in the field of stuttering. The author has gleaned wisely and with a generous lack of prejudice, and has supplemented his gleanings with much practical material from his own experience.

This reviewer gets many inquiries from parents and teachers for practical help in the form of literature that may be read by the lay person. At last I have found what I am looking for to which to refer these inquirers. I can recommend this book for this purpose with confidence.

ROBERT WEST.

University of Wisconsin

MURRAY, ELWOOD. The Speech Personality. Textbook and guidance manual of individual and class projects. New York: J. B. Lippincott Co. Revised edition, 1944. Pp. xii +565.

Speech teachers and speech correction workers who recognize the importance of basing corrective measures on a broad base of knowledge and principle will welcome this revised and supplemented edition of Professor Murray's well established and widely used textbook. To Murray must go a major share of the credit for making the term *speech personality* meaningful and significant in present-day speech education. The term takes on added implications and even greater significance from the supplementary material contained in this new edition of *The Speech Personality.*

Readers who are familiar with the earlier editions are already acquainted with Professor Murray's basic point of view. Emphasis is placed on the relationship of speech to those other aspects of behavior which are subsumed under the general heading of personality. The teaching principles and techniques presented by Murray are designed not merely to develop speech skills in the traditional sense, but also to promote personality adequacy. Speech occurs in social situations, and it is Murray's contention that it should serve to bring about integrative and constructive human relationships. He holds this point of view not only with regard to ordinary speech education but also with regard to speech correction, stressing more than is customary the importance of personality re-education in clinical speech work.

The major addition to the book as it appears in the new edition is a 41-page supplement entitled "Speech Development Through the Improvement of the Evaluative Reactions: Supplementary Methods from General Semantics." A 30-page exposition of the major principles and techniques of general semantics, as these apply to speech education, is followed by a series of assignments and readings which can be used to apply general semantics in the speech classroom. This sort of material makes increasingly meaningful the implications of general semantics and the practical respects in which it tends to depart from traditional teaching procedures and aims. Teachers who have used Murray's text in the past will want to change now to this new edition in order to take advantage of this important supplementary material, and many who have not used it will want to consider anew the classroom value of *The Speech Personality* in view of the new material which this edition contains.

The value of the book to speech correctionists lies in the theoretical emphasis which it places on personality adjustment in relation to speech training, and in the practical means which it provides for personality re-education in connection with speech correction.

WENDELL JOHNSON

State University of Iowa